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FURTHER OBSERVATIONS ON CHRONIC NEPHRITIS AND LEAD POISONING IN QUEENSLAND, WITH COMMENTS ON THE FEDERAL OFFICIAL INQUIRY.[1]

By L. J. JARVIS NYE, M.B., Ch.M. (Sydney),
Brisbane.

RARELY is the truth of a medical thesis established without the expenditure of great labour and the passage of much time. The researchers must examine so many side tracks that progress to final conclusions and acceptance is extremely slow. The case of lead poisoning and chronic nephritis in Queensland is no exception to the general rule.

It has been known for many years that chronic nephritis in young people in Queensland is extremely common. It is questionable, however, if anyone prior to 1928 realized the amazing amount of morbidity due to nephritis. Since the actual numbers seen by any one individual are comparatively few, only a State-wide survey based on data collected from all sources could reveal the real ravages of the disease. In that year Croll[1] and I[2] published the figures showing the deaths from chronic nephritis in age groups up to forty years of age in Queensland, New South Wales, Victoria, South Australia and New Zealand. (It is unfortunate that no figures were available from Western Australia.)

A glance at these figures showed clearly that the increase in the death rate from chronic nephritis in young people in Queensland was a tragic reality which presented an important field for research. It was noted that in Queensland 1,250 persons died from the disease during the years 1917 to 1926 and only 242 deaths were recorded in South Australia during the same period. Whilst the three States,

[1] Abstract of a special report to the Chronic Nephritis Subcommittee of the Queensland Branch of the British Medical Association.

New South Wales, Victoria and South Australia had a death rate per 100,000 of approximately 100, that for Queensland was more than three times as great.

Since 1928 I have contributed further articles[3][4] on the subject, bringing forward new evidence to indicate that lead poisoning in childhood plays at least a part in causing this increased mortality. New interest was immediately stimulated in an old subject, for as early as 1892 Turner and Gibson[5] had published numerous cases of lead poisoning.

At the request of the Queensland Branch of the British Medical Association, the Federal authorities agreed to investigate the question. They appear to have prosecuted their search with much vigour and have collected a mass af valuable data, a copy of which was made available to the Queensland Branch of the British Medical Association by courtesy of Dr. R. W. Cilento, while a *précis* of the findings was published in *Health*, in December, 1932.

The contention that kidney disease in young people in Queensland is extraordinarily prevalent has been upheld; and that in some way lead is involved in the renal syndrome is also maintained. It is, however, greatly regretted that the inquiry was terminated before any final conclusions were arrived at. The report admits the incompleteness of the research along certain lines and the *interim* nature of its conclusions.

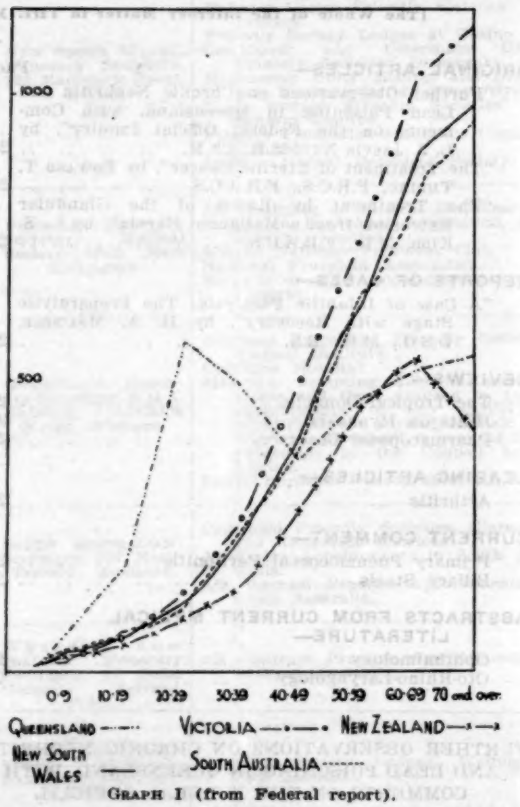
The present communication contains new evidence on a study of 186 patients with chronic nephritis (under forty years of age) and the avenues of research which the Federal report suggested as being necessary to the elucidation of the problem have been followed to their logical conclusion by the collection of the necessary statistical and clinical evidence. The willing cooperation of the public bodies and individuals who were asked to furnish figures and facts is indicative of the widespread interest which is taken in the subject. In making a study of the mortality from chronic nephritis in all ages in Queensland and a comparison with the figures for the other States, Croll pointed out the interesting fact that, although the death rate in Queensland is about three times as great as in the other States for ages under forty years, after forty years of age the deaths registered under chronic nephritis are considerably less than in the southern States.

This is so evident (see Graph I) that it has called forth the following statement in the Federal report.

> There is a very considerable increase in the death rates of persons under forty years from chronic nephritis in Queensland as compared with other States; there was, nevertheless, an equally marked drop as compared with other States in the death rate from forty years onward. If the figures for the Eastern States are taken as normal whatever cause is acting in Queensland would appear to act, not as a separate or independent factor but as something which accelerates by some twenty years the deaths of a certain number who would normally die of chronic nephritis.

This suggests that there is possibly some unknown effect of a tropical climate or some toxin operating on those persons with an arteriosclerotic diathesis thereby causing this premature vascular sclerosis.

In spite of these figures one's observations make one doubt this contention, for it appears to us that chronic nephritis after forty years of age is just as commonly seen in Queensland as elsewhere. Chronic nephritis is so closely allied with other vascular diseases after forty years of age that it is almost a matter of coincidence whether they are called arteriosclerosis, hypertension *et cetera* or chronic nephritis, and in order to make a more accurate survey of the question, it was

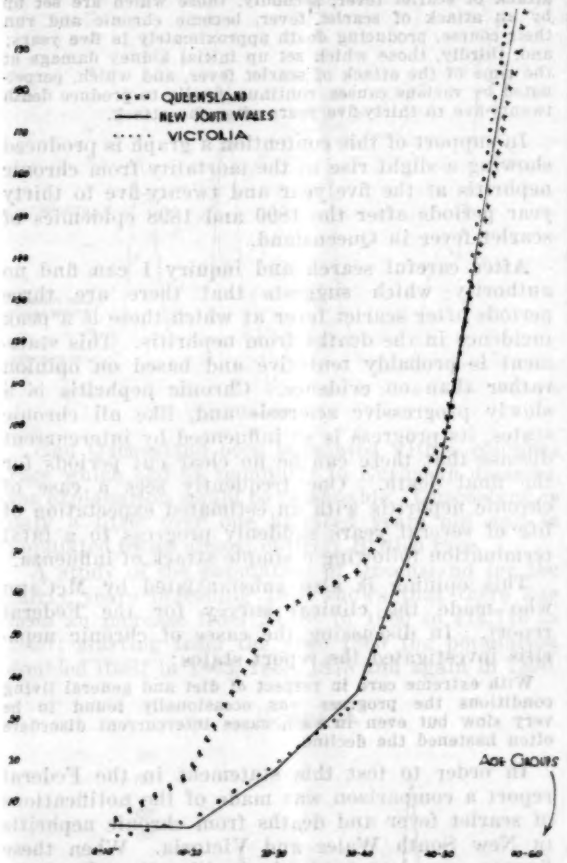


GRAPH I (from Federal report).

decided to obtain the figures for all diseases which might be classified in the same group as chronic nephritis.

Figures were then obtained for Queensland, New South Wales and Victoria showing the total deaths classified under chronic nephritis, uræmia, hypertension, cerebral hæmorrhage and thrombosis and arteriosclerosis. When a comparison is made of these vascular diseases (see Graph II) it is found that the figures for Queensland show the same greater incidence in the groups under forty years of age, but that after forty there is comparatively little difference between the three States. It is difficult to explain why so many medical men in New South Wales and Victoria should sign many death certificates as chronic nephritis while those of Queensland more frequently diagnose some

vascular sclerosis. At least one must agree that if chronic nephritis is less common in Queensland than in the other States, vascular scleroses are more common. This may in part be due to the explanation put forward by Croll that in cooler climates there is more actual nephritis in older people than in the warmer climate of Queensland. As the problem under review in Queensland is nephritis



GRAPH II.

of the diffuse vascular sclerotic type, by making this comparison of nephro-vascular diseases one is reasonably safe in stating that there is some factor operating which adds large numbers of deaths to the absolute total of the series and not as suggested in the Federal report

> some local factor that culls those persons liable to death from chronic nephritis at an age considerably earlier than they would otherwise die.

Pathogenesis.

In discussions on the ætiology of nephritis, scarlet fever usually occupies a prominent place and in the Federal report many authorities are quoted to show that nephritis is a common complication of the disease, while certain suggestive deductions are drawn from statistical evidence as will be described later.

In order to understand the question, it must be realized that there are two varieties of nephritis for which this *Streptococcus scarlatinæ* is responsible. The first is the non-embolic focal glomerulonephritis which occurs in the early stages of the disease. In this case the damage is confined to a limited number of glomeruli and it is therefore characterized by absence of œdema, no increase in blood pressure and little or no renal insufficiency. Being a benign condition, its course is entirely dependent upon the disease which it complicates. This benign type must be carefully distinguished from the second variety, a diffuse glomerulonephritis which occurs later in the convalescence and usually manifests itself on or about the nineteenth day of the disease. This late appearing diffuse nephritis is the variety which may develop into the chronic form.

The incidence of nephritis in scarlet fever varies in different countries. Elwyn[6] quotes Rolley as finding this complication in 7% of his patients, Pospischil and Weiss in 9%, Heubner in 10% to 20%, and Jockmann in 20%.

The records of the Infectious Diseases Hospital at Fairfield, Melbourne, show the incidence of nephritis in scarlet fever cases from 1920 to 1931 to be only 0·75%; of these it is known that the majority represents cases of focal nephritis which clear up completely before the patient has left hospital. Scholes,[7] the Medical Superintendent, who probably has had more practical experience in scarlet fever than any other man in Australia, states that he does not believe there is any evidence of the tendency for scarlet fever patients to develop nephritis in later life if they have not developed nephritis with scarlet fever. He considers the severity and duration of the attack to be the main factor in the prognosis and even when severe nephritis develops, the majority of cases completely clear up later on.

Kaye,[8] in the study of nephritis at the Alfred Hospital, found that 250 patients with fatal chronic nephritis were admitted during a ten year period; of these only four gave a history of scarlet fever in youth or infancy. As these patients were aged sixty-eight, sixty-five, sixty-two and sixty-one years at the time of death, it is not probable that scarlet fever played much part in the production of their fatal nephritis. Some of these patients were so nearly dead that a reliable history could not be obtained. He therefore studied a further series of 157 patients with chronic nephritis and found that scarlet fever had occurred in youth or infancy in twelve instances, while an infectious disease which was possibly scarlet fever had occurred in three more. The ages of the scarlatinal patients when treated were seventy-eight, seventy-two, sixty-five, sixty-six, fifty-four, forty-two, thirty-four, twenty-eight, twenty-six, twenty-two and twenty; the ages of the patients in the doubtful cases were sixty-five, thirty-two and thirteen. It is reasonable to regard scarlet fever in childhood as insignificant where the patient is found to have chronic nephritis at an age above fifty. Disregarding patients

over this age, he therefore found in his series of 157 nephritics, six definite and two indefinite cases of scarlet fever or 5%. This percentage is probably no greater than would be found in a series of healthy controls.

I have personally made follow up examinations of 43 cases of scarlet fever in Queensland and in not one instance have I found chronic nephritis developing later in life, although several patients had had albuminuria during the febrile stage of the disease.

In 186 cases of chronic nephritis which have been investigated in this series, there were only two patients who gave a history of acute nephritis or hæmaturia at any period in their childhood. In these enlightened days of free medical service it is hardly conceivable that many children would have an attack of acute nephritis and remain untreated, for such objective signs as œdema and hæmaturia, which are so usually present, could hardly pass unobserved.

This evidence tends to suggest that the type of nephritis under review does not originate in an attack of acute diffuse nephritis and progress to the chronic state in which it is observed. It rather suggests a slowly progressive diffuse sclerosis extending over many years such as would be produced by lead.

It is well known that in the tropics scarlet fever and in fact all the exanthemata are sometimes so mild as to pass unrecognized and, although Herringham[9] states that he has observed nephritis in patients who have merely been exposed to scarlet fever infection, nevertheless it is probable that the toxins associated with mild scarlet fever are not so liable to cause nephritis as those of a more virulent variety.

A review of the incidence of scarlet fever in Queensland and a comparison with other States is of interest and bears out the commonly recognized fact that scarlet fever is relatively less common in tropical climates.

Although these figures show conclusively that scarlet fever is far less commonly reported in Queensland than in the other States, it may perhaps be argued that, even if it is less frequently notified, it may be of a more virulent character; this is definitely not the case. The percentage mortality from scarlet fever is as follows:

New South Wales	1·12%
Victoria	0·75%
Queensland	1·09%
South Australia	0·75%
New Zealand	0·93%

A very interesting comparison can be drawn between the figures for nephritis and scarlet fever in New Zealand, a cold climate, and Queensland with its tropical climate. It will be noted that the notifications of scarlet fever during the past ten years in New Zealand were 14,987 and in Queensland only 4,098. On the other hand the deaths from nephritis in the ten to forty age groups during the same period in New Zealand were 436, while in Queensland the number was 1,299.

When discussing scarlet fever as a possible ætiological factor, the Federal report states:

> When, however, the deaths from scarlet fever during the intense epidemics of 1890 and 1898 are considered in reference to the death rates for nephritis over the period 1890 to 1930, definite impressions appear to be made at three periods at which nephritis manifests itself in relation to scarlet fever; that is to say, firstly those cases which occur as a fatal nephritis complicating the attack of scarlet fever, secondly, those which are set up by an attack of scarlet fever, become chronic and run their course, producing death approximately in five years; and, thirdly, those which set up initial kidney damage at the time of the attack of scarlet fever, and which, perpetuated by various causes, continue, finally to produce death twenty-five to thirty-five years after the attack.

In support of this contention a graph is produced showing a slight rise in the mortality from chronic nephritis at the five year and twenty-five to thirty year periods after the 1890 and 1898 epidemics of scarlet fever in Queensland.

After careful search and inquiry I can find no authority which suggests that there are three periods after scarlet fever at which there is a peak incidence in the deaths from nephritis. This statement is probably tentative and based on opinion rather than on evidence. Chronic nephritis is a slowly progressive sclerosis and, like all chronic states, its progress is so influenced by intercurrent disease that there can be no clear cut periods for the final death. One frequently sees a case of chronic nephritis with an estimated expectation of life of several years suddenly progress to a fatal termination following a simple attack of influenza.

This opinion is also substantiated by McCann who made the clinical survey for the Federal report. In discussing the cases of chronic nephritis investigated the report states:

> With extreme care in respect of diet and general living conditions the progress was occasionally found to be very slow but even in such cases intercurrent disorders often hastened the decline.

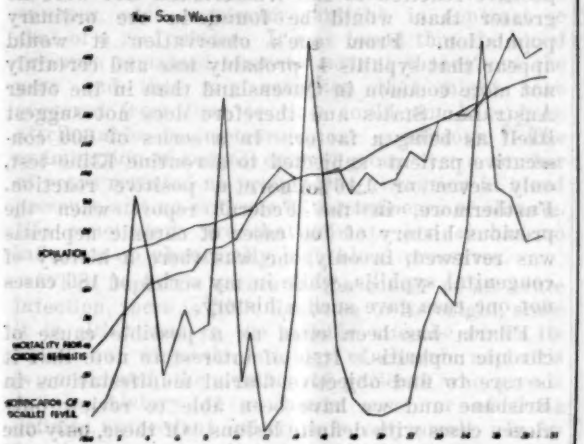
In order to test this statement in the Federal report a comparison was made of the notifications of scarlet fever and deaths from chronic nephritis in New South Wales and Victoria. When these figures are plotted together with a graph of the population there is a clear demonstration that the deaths from chronic nephritis in these States bear little or no relation to the epidemics of scarlet fever.

Graphs III and IV show that the yearly mortality rises gradually in direct proportion to the increase in population, and although in New South Wales and Victoria 1903 and 1909 were big epidemic years for scarlet fever, there are no peak years for deaths from chronic nephritis.

The Federal report gives as a possible explanation of the fact that no similar series of figures are recorded for other States that:

> These (States) had their epidemic incidences of scarlet fever to a degree comparable with the epidemics in Queensland (1890 and 1898) at a period ten to twenty years earlier and had already passed at the period of review from a stage of mass susceptibility into the present more general phase of adult mass immunity.

This suggests that in the years 1890 and 1898 Queensland was in a state of mass susceptibility. It is very difficult to conceive how the young State of Queensland could be in a different state of susceptibility from that of New South Wales or Victoria.



GRAPH III.

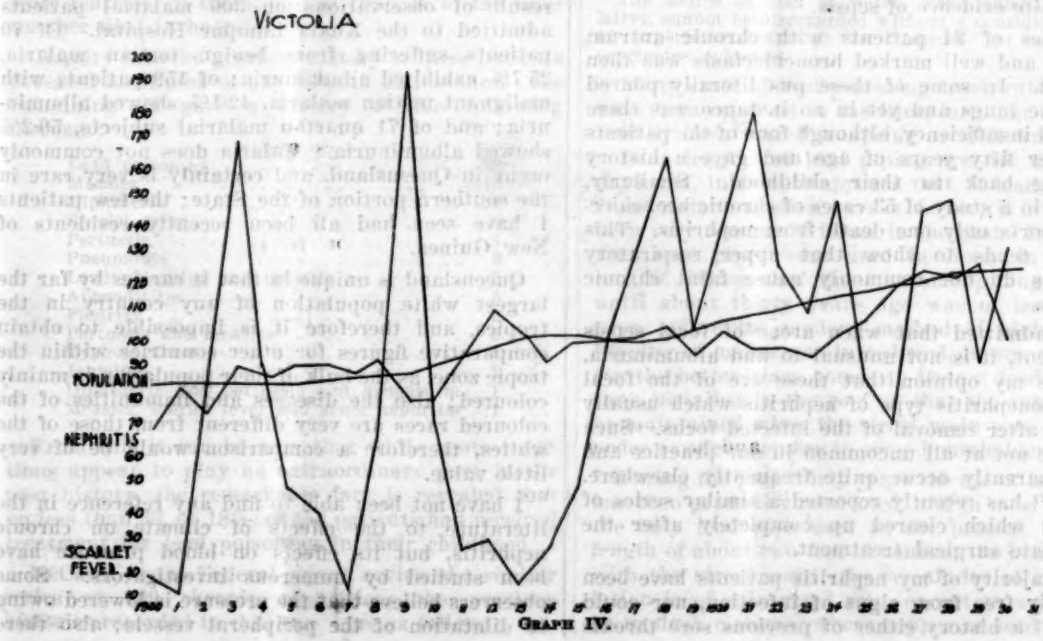
Had Queensland been populated with a coloured race which had no immunity to European diseases, the explanation would be reasonable. Queensland is only a little over one hundred years old and is populated by an almost entirely British race.

A study of the population of Queensland for the thirty years preceding 1890 shows that there has been an increase from 25,788 in 1860 to 414,716 in 1890; starting from the year 1860 the population doubled itself in 1863, 1868, 1878 and again in 1890. This increase is made up largely by immigrants from other States and from overseas, for during this period there was an average number of immigrants to Queensland of approximately 21,000 persons *per annum*. Furthermore it is found that scarlet fever was by no means new to Queensland before 1890. Unfortunately the notifications of the disease before that year are not available; the deaths were as shown in Table I. This shows that although 1890 was one of the epidemic years referred to, the deaths were 61 when the population of Queensland was 414,716, while in 1875 it will be noted that the deaths were 51 when the population was only 172,402. This suggests that there was a severe epidemic in that year extending to 1876 when the deaths were 28. There is also evidence that cases of scarlet fever have occurred almost continuously since 1872, for in nearly every year deaths

TABLE I.

Showing Deaths from Scarlet Fever Registered in Queensland.

Year.	Males.	Females.	Persons.
1872	2	10	12
1873	—	4	4
1874	3	7	10
1875	21	30	51
1876	12	16	28
1877	1	4	5
1878	2	2	4
1879	—	—	—
1880	—	—	—
1881	1	—	1
1882	—	2	2
1883	2	1	3
1884	4	4	8
1885	—	3	3
1886	1	—	1
1887	—	1	1
1888	—	3	3
1889	—	1	1
1890	26	35	61



GRAPH IV.

have been recorded. It therefore appears that the susceptibility or immunity of the population of Queensland must be very similar to that of the other Australian States.

From an impartial survey of the above evidence one must conclude that scarlet fever plays a very doubtful if not a negligible part in the production of fatal chronic nephritis and it certainly does not appear to be the agent which is responsible for the increased incidence of nephritis in Queensland.

In measles and diphtheria, kidney disease is usually a rare complication and in my observations I could find nothing to suggest any association between these diseases and chronic nephritis in Queensland.

Chronic tonsillar infection is considered by some to be a frequent cause of nephritis. Alport and Peterson and Wyllie[10] have shown that acute hæmorrhagic nephritis in children is frequently associated with tonsillitis and similar conditions such as adenitis or *otitis media*, the causal organism usually being the streptococcus, although the staphylococcus and pneumococcus are sometimes found. To investigate this possibility 42 patients suffering from chronic nephritis were carefully studied with a view to ascertaining the presence of some tonsillar or other upper respiratory infection. In fifteen there was no sign of such infection and the tonsils could be considered quite healthy, thirteen had had their tonsils and adenoids removed in childhood and there was no other infection present. Ten had definitely infected tonsils. This survey corresponded very closely with a control series of 42 healthy young adults in whom eighteen showed no signs of infection, nine had had their tonsils removed, while fifteen had definite evidence of sepsis.

A series of 24 patients with chronic antrum infection and well marked bronchiectasis was then examined. In some of these pus literally poured out of the lungs and yet in no instance was there any renal insufficiency, although four of the patients were over fifty years of age and gave a history extending back to their childhood. Similarly, Mett,[11] in a study of 53 cases of chronic bronchiectasis reports only one death from nephritis. This evidence tends to show that upper respiratory infections do not commonly cause fatal chronic nephritis.

It is admitted that when areas of focal sepsis are present, it is not unusual to find albuminuria, but it is my opinion that these are of the focal glomerulonephritis type of nephritis which usually clear up after removal of the infected focus. Such cases are not at all uncommon in our practice and they apparently occur quite frequently elsewhere. Alport[12] has recently reported a similar series of 14 cases which cleared up completely after the appropriate surgical treatment.

The majority of my nephritis patients have been singularly free from signs of infection, nor could one elicit a history either of previous sore throats or pyelitis or of anything to suggest periodic bouts of pyrexia.

The Federal report suggests "damage produced by congenital syphilis" as being a possible cause. The number of our nephritic patients who gave a positive reaction to the Wassermann test were no greater than would be found in the ordinary population. From one's observation it would appear that syphilis is probably less and certainly not more common in Queensland than in the other Australian States and therefore does not suggest itself as being a factor. In a series of 600 consecutive patients subjected to a routine Kline test, only seven or 1·16% gave a positive reaction. Furthermore, in the Federal report when the previous history of 100 cases of chronic nephritis was reviewed, in only one was there a history of congenital syphilis, while in my series of 186 cases not one case gave such a history.

Filaria has been cited as a possible cause of chronic nephritis. It is of interest to note that it is rare to find objective filarial manifestations in Brisbane and we have been able to review only eleven cases with definite lesions. Of these, only one patient showed evidence of chronic nephritis, and as she was sixty-five years of age, it is improbable that filaria was the responsible agent. If such were the case, it would be reasonable to assume that many more hosts of such infestation would be the victims of chronic vascular sclerosis, for it would suggest that the parasite produced some specific sclerosing toxin.

Cilento[13] states that nephritis is very common amongst the natives in the South Sea islands. It is possible that malaria may be an ætiological factor amongst these people, for Green[14] records the result of observations on 300 malarial patients admitted to the Kuala Lumpur Hospital. Of 70 patients suffering from benign tertian malaria, 25·7% exhibited albuminuria; of 159 patients with malignant tertian malaria, 42·1% showed albuminuria; and of 71 quartan malarial subjects, 59·2% showed albuminuria. Malaria does not commonly occur in Queensland, and certainly is very rare in the southern portion of the State; the few patients I have seen had all been recently residents of New Guinea.

Queensland is unique in that it carries by far the largest white population of any country in the tropics, and therefore it is impossible to obtain comparative figures for other countries within the tropic zone, as the bulk of their population is mainly coloured; also the diseases and immunities of the coloured races are very different from those of the whites, therefore a comparison would be of very little value.

I have not been able to find any reference in the literature to the effects of climate on chronic nephritis, but its effects on blood pressure have been studied by numerous investigators. Some observers believe that the pressure is lowered owing to dilatation of the peripheral vessels; also there

is less vascular sclerosis on account of the fact that in tropics people do not work so hard.

Lemann,[15] working in New Orleans, has recently made a study of the effects of a hot climate on 45 persons. The climate of New Orleans provided an opportunity to study the effects of a long, hot summer, with maximum sunlight, in contrast to that of a real winter. It was found that the blood pressure remained almost constant throughout the year and that any variation that might have occurred from time to time could not have been attributable to meteorological conditions. The examination of a small control group over a number of years revealed no pronounced alteration in blood pressure and no evidence of the influence of climate. His conclusions are that climate *per se* has no influence on abnormally high blood pressures.

When nephritis occurs in the course of an acute infection, there is little doubt as to its origin, since it is either due to the specific microbe or to its toxin; but when the ætiology of chronic nephritis is considered, the number of patients who give a definite history of an acute onset or of some definite antecedent infection is very small. In by far the greater number the condition begins insidiously and is discovered by accident; nevertheless careful inquiry shows that symptoms had already existed for a considerable time. In the Federal report, McCann, in his investigation of 100 chronic nephritics under forty years, found that "65% gave a history of slow onset after a delicate childhood".

In order to search for some possible ætiological factor, 186 cases were collected from the Brisbane General Hospital records and from my private and out-patient practice during the past six years. Amongst these patients, 84 were males and 102 were females; of these, no less than 47 of the total number died in hospital.

Inquiry into the previous history of these patients revealed no illness of any note in 92 instances; the remaining 94 gave a history of the following diseases:

Lead poisoning	28
Measles	18
Diphtheria	9
Scarlet fever	9
Pertussis	7
Pneumonia	6
Lead poisoning and measles	5
Appendicitis	3
Pleurisy	3
Pertussis and measles	2
Typhoid fever	1
Jaundice	1
Diphtheria and pertussis and hæmaturia	1
Measles, scarlet fever and acute nephritis	1

From this it can be seen that while acute infections appear to play no extraordinary part in the past history, the remarkable fact is revealed that no less than 33, or 18%, of the patients had received treatment for lead poisoning in their childhood.

McCann, in the Federal report, reviewed a similar series of 100 chronic nephritics; the previous histories recorded in this case were as follows:

Plumbism, definite	30
Plumbism, indefinite	16
Measles	14
Pertussis	11
Diphtheria	9
Typhoid fever	5
Rheumatic fever	3
Scarlet fever	4
Acute nephritis	3
Eclampsia	2
Pneumonia	2
Congenital syphilis	1

The report states that:

> Of all etiological factors investigated, none was so commonly suggested as plumbism amongst 100 cases of the Brisbane series, 46 per cent. stating that they had been treated at an earlier period for lead poisoning. In 30 of these cases hospital records indicated typical plumbism and in 16 indefinite signs.

That 46% of a series of 100 chronic nephritics should give a history of plumbism was surely evidence of extraordinary value.

In my series of 87 patients who were interviewed, 74 had spent their childhood in Queensland, and in 71 instances there was a definite history that the paint on the verandas had been dry and powdery. Forty-six were nail-biters or thumb-suckers, and in seven cases the parents gave the history that the child had been in the habit of licking the rain drops from the veranda railings. In the Federal series 63% "definitely were now or in early life had been nail-biters". This information shows that the majority had been exposed to the risk of lead poisoning.

Sources of Lead.

There are several possible sources of lead, the most frequent probably being from water supply. The Federal report states:

> The source of lead in water, while admittedly speculative, cannot be disregarded without a considerable range of experiments difficult to institute but essential to conclusive evidence.

Many cases of lead poisoning from water supplies have been reported in the literature, but these have usually been traced to spring or well water.

Long lengths of lead piping are not used nowadays and, in fact, it appears that, if lead poisoning from water supplies were a factor, it would have been very commonly found in the past. It is known that lead pipes were used in Rome in the year A.D. 20. Since that time the only piping available until about thirty years ago was of lead. Moreover, before the modern method of high pressure tapping of mains was introduced, it was customary for the better class homes to have a lead reservoir tank installed in the roof so that the supply could be maintained when the street main was cut off, in order to effect repairs to some householder's supply.

During the past thirty years the use of lead piping has been gradually restricted, and in most instances the only lead pipe used at the present time is a length of about two feet to connect the house supply with the street main; the remainder of the installation is of galvanized iron, except to wash basins or to flush cisterns, where copper or lead is used.

In some districts where electrolysis is highly prevalent, a lead service pipe from the main to the building line is still used for a distance averaging about thirty feet.

Since it is admitted that a small amount of lead piping is used in Queensland, it would be well to study the plumbisolvent qualities of water in general and to survey the Brisbane water supply in particular.

The causes of lead solvency in water are high carbon dioxide content, humic, peaty and mineral acids; therefore, in investigating the question, it is very necessary to study the source of the supply.

The pH of a water is the measure of its effective acidity or alkalinity; pH values below 7 denote an acid water, above 7 an alkaline water. The hydrogen ion concentration of a water is the controlling factor as far as its corrosive and plumbisolvent qualities are concerned, and in modern water supplies the pH is readily and inexpensively corrected to any value considered desirable by adding the adequate amount of alkali to the supply. It has been definitely proved that alkaline waters do not offend in the matter of lead solvency, and many waters need no correction. Brisbane River water is a hard, alkaline water, but the Enoggera supply is soft and frequently acid. On this account it is now treated with hydrated lime to minimize corrosion in the mains and to overcome its tendency to dissolve lead.

A pH value of 7·05 and under must be regarded with suspicion, while a reading of under 6·5 points to its ability to dissolve lead in quantities too large to be safe for human consumption.

In the Enoggera supply the alkalinity of the water is now maintained at a pH value of not under 7·2.

Brisbane's Water Supply.

The Enoggera Creek was the first of the sources of supply to Brisbane, and the Enoggera reservoir was completed in 1866. This supply was augmented by water from Gold Creek in 1886. Both of these are soft water.

The hard (alkaline) Brisbane River water was first made available in 1893, in conjunction with Gold Creek water, and, no doubt, had the effect of neutralizing the soft water from Gold Creek. According to Bush,[16] it can be definitely stated that the area supplied by Enoggera (soft water) was restricted from that year (1893) to Brisbane city and portion of Windsor, Hamilton, Newstead, Ithaca and Toowong.

In 1912 the supply from Enoggera was further restricted by the construction of the filtration plant as far as it related to the area served, but, of course, the population of those districts has increased. The districts supplied by Enoggera since 1912 are Ashgrove, the lower portions of Rosalie, portions of Ithaca, Rainworth, Newmarket, Windsor, Milton Road and Roma Street, City.

It will therefore be seen that since 1892 only a very limited area of Brisbane has been supplied with soft, plumbisolvent water, and for the past twenty years that area has been still further reduced.

Although it is admitted that acid conditions have been present on occasions in this limited area supplied by the Enoggera water, Chamberlain[17] states that recently he had the opportunity of examining a twelve inch main which had carried Enoggera water for over fifty years; many small reticulation mains had been in service on this supply for a still longer period. It was his opinion that if the water was sufficiently acid to be plumbisolvent, the iron mains would have corroded and certainly would not have given such long service. Moreover, he states that a condition termed "red water" is commonly found in acid waters, but this has not been observed in the case of Enoggera.

Although the foregoing facts appear to be sufficiently convincing, it was thought advisable to inquire into this question still further. For this purpose a study was made of a map of Brisbane published by Gutheridge[18] in 1928, showing the areas supplied from the various reservoirs. A series of twenty consecutive cases of definite plumbism with paralysis was then selected. The district in which each patient lived when the disease was acquired was plotted on a map of Brisbane showing the area supplied by soft water. Reference to such a map (Figure I) shows that the cases bear no possible relation to town water supply.

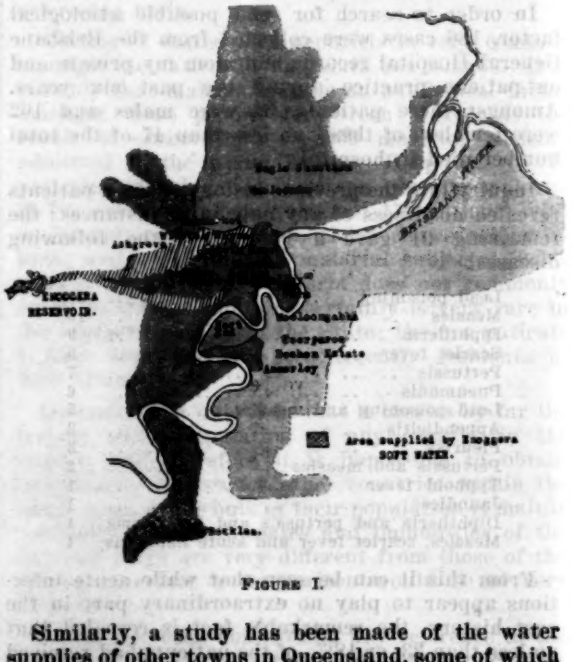


FIGURE I.

Similarly, a study has been made of the water supplies of other towns in Queensland, some of which have been especially analysed for this investigation. In no case did one find any evidence to suggest plumbisolvent qualities in the water with associated plumbism or nephritis. The cases of Toowoomba

and Charters Towers are of especial interest in supplying negative evidence. In Toowoomba the mortality from chronic nephritis is low, and yet the water is obtained from wells and is definitely plumbisolvent, its pH ranging from 6·5 to 7·0. In Charters Towers, where the mortality from chronic nephritis is nearly as great as in Brisbane, the water is not plumbisolvent. It is very alkaline and has a pH of 7·45 (one sample). In Rockhampton, where the mortality from chronic nephritis is also great, the water is again extremely alkaline, the pH value varying from 7·8 to 8·6.

From the above evidence it appears that lead from town water supplies plays a negligible part in the causation of plumbism in Queensland.

Galvanized iron tanks have been used in the past to supply most of the people in Queensland with water for drinking purposes, and it has been suggested that the lead is obtained from this source owing to the fact that a great deal of solder is used in the ordinary galvanized iron tanks. This contention has been disproved by workers who have shown that metallic zinc, which is always present in these tanks, will combine with the lead carbonate, forming zinc carbonate, thereby precipitating metallic lead, which, of course, is insoluble.

Further evidence against an origin from this source is that in the country areas lead poisoning is almost unknown, and yet the only source of drinking water is from galvanized iron tanks.

Final evidence against all water supplies is the reasonable assumption that if this were the source, all members of the family or community would be similarly and simultaneously affected, whereas the only sufferers are a selected group of children, chiefly those between the ages of one and a half and three years, who are nail-biters or finger-suckers, as described later.

The question of the spraying of vegetables with lead arsenite has been raised, but this can be dismissed, as it has been used only during the past fifteen years, and only intensively during the past nine years, whereas lead poisoning has been recorded for the past forty years.

The only probable source is, as suggested by Gibson, the lead paint on the dwellings. The lead carbonate which is powdered by the tropical sun is removed from the walls and railings by contact with the hands, which become sticky with saliva, and since the fingers are frequently in contact with the mouth, a small amount of lead carbonate is being constantly transferred to the mouth and ingested. Another very common factor is the habit amongst children of licking the droplets of rain from the veranda railings. This information was volunteered by some of our patients, who stated that it was a favourite pastime of their childhood.

It has been previously shown that plumbism affects only a selected group of young children and chiefly those who have these habits which must be considered definite localizing factors suggesting that the lead is ingested from the finger tips. The age of onset of lead poisoning surely suggests some factor which is only accessible to young children.

The Federal report states that:

> In 66% of the cases quoted the illness commenced at four years or under, and closer investigation only served to reduce the average age of onset to from one and a half to three years of age.

This is the age when children would be most frequently and continuously supporting themselves on the varanda railings. When it is known that these railings are covered with powdered lead carbonate and the children's fingers are in contact with it hour after hour, day after day, month after month, year after year, is there any reasonable need to search further for the *fons et origo*?

Chronic Nephritis as a Sequela of Plumbism.

In order to ascertain whether many of our patients with infantile plumbism developed chronic nephritis in later life, a series of thirty-four cases of severe lead poisoning with paralysis was studied. Inquiry revealed that they had all spent their childhood in wooden houses in contact with chalky paint; eighteen were nail-biters, eight were both nail-biters and thumb-suckers, and four were thumb-suckers, while only four gave a history of no such habits. In all, poor physical development was a constant feature, and in five instances other members of the family had been affected with the same disease.

It was noted that in seven instances no albumin was present in the urine; nevertheless the results of the renal function test revealed the appalling fact that of these thirty-four unfortunate young people, twenty-nine had renal insufficiency definitely established.

The Federal Commission making a similar review examined twenty-eight severe and typical cases of plumbism in childhood, and the report states:

> In 19 of these chronic nephritis was found at varying inetrvals after the recorded plumbism with paralysis. There was thus demonstrated here, as elsewhere, a definite relationship between the occurrence of marked plumbism and the subsequent occurrence of chronic nephritis in these cases.

The Federal report also states that:

> Of those considered to be definite cases of plumbism, 78% were nail-biters, and in eight cases there was a definite history of persistent thumb-sucking.

As these were all cases of severe plumbism with paralysis, it is an easy matter to follow the damaging effects on the organism generally, but there must of necessity be a great many cases which have escaped recognition, since the effects are not manifest sufficiently to produce the well known signs and symptoms of the disease. Hence it follows that when nephritis is discovered late in life, the association with plumbism is not suspected.

Chalking of Paint.

In order to obtain practical proof of the effect of climate on paint, information was obtained from the Chief Engineer of the Queensland Railways as to factors which were mainly responsible for the

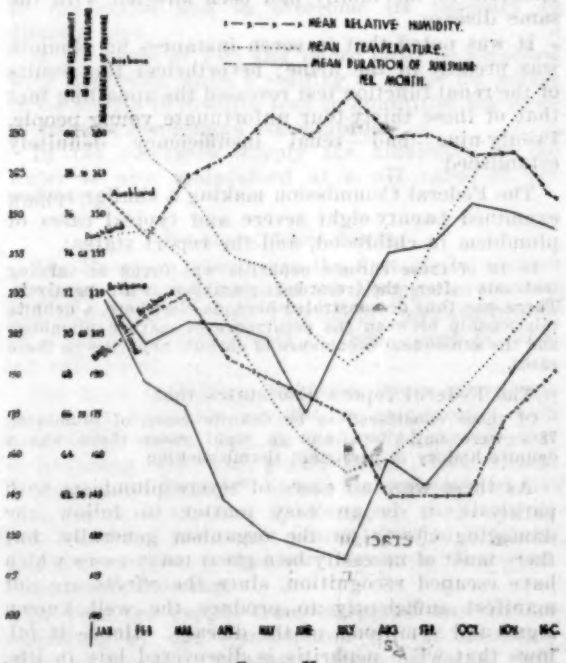
drying of paint. Queensland covers a very vast area in tropic and subtropic climates, and it appeared that the Maintenance Engineers' Department should be in a position to supply very accurate and useful information. The results of their observations are that the chief factors in deterioration of paint are heat, long hours of sunshine and high wind velocity, while humidity has a slight protective action.

Type of House.

The Federal report, in making a comparison between Queensland and New Zealand, states:

> In respect of the further contention that the important factor is not the matter of residence in a painted wooden house, but certain climatological factors affecting the weathering of paint, examinations in this regard were instituted. From these it became apparent that in New Zealand, which, as stated, with the highest percentage of painted wooden houses in Australasia, has the lowest percentage of chronic nephritis and no lead poisoning, the lead paint nevertheless does "weather", and soluble lead in proportions and quantities similar to those found in Queensland is present on the walls, veranda rails, and so forth, of houses.

Graph V shows a comparison of the various climatological factors which are mainly responsible for desiccation of paint, namely, heat, duration of sunshine, while humidity has a slight protective action. It will be noted that in Brisbane the duration of sunshine is nearly twice as great as in Auckland, and there is considerable difference in the temperature throughout the year. If this difference is so great in the case of Auckland, how much greater must it be in New Zealand as a whole? The wind velocity being slightly greater in Brisbane is an additional destructive factor, while the greater humidity in Auckland would tend to act as a protective.



GRAPH V.

Apart from these factors which are responsible for the powdering of paint in Queensland, the homes are almost invariably provided with verandas, sometimes on all sides. In New Zealand such verandas are rarely, if ever, seen. The following is an extract from the report written by the Government Statistician of the Dominion on this subject.

> It is not usual to find houses of the type built in Queensland. Generally speaking, houses are not built nowadays in New Zealand with verandas at all, and certainly very few where a gate is required to make them safe for children to play upon. The small sun porch, rather than anything in the way of a large veranda, is the fashion at the present time.

Similar arguments may be raised in the case of the other Australian States, for it is obvious that paint powders anywhere and children are placed on verandas. The whole question obviously depends on the quantity of lead intake, and this in turn depends on the amount of lead available and the duration of access. In cooler climates children are often confined to verandas for short periods, but are not allowed to remain long, owing to the risk of catching a chill. In Queensland, owing to the hot climate extending over eight or nine months of the year, children are kept on the verandas for long periods day after day, and it is this minimal ingestion of lead over long periods which is responsible for the pathological changes. It is also possible, as previously suggested, that the effects of a hot, humid climate may be a factor in increasing the susceptibility to lead.

Distribution of Chronic Nephritis in Queensland.

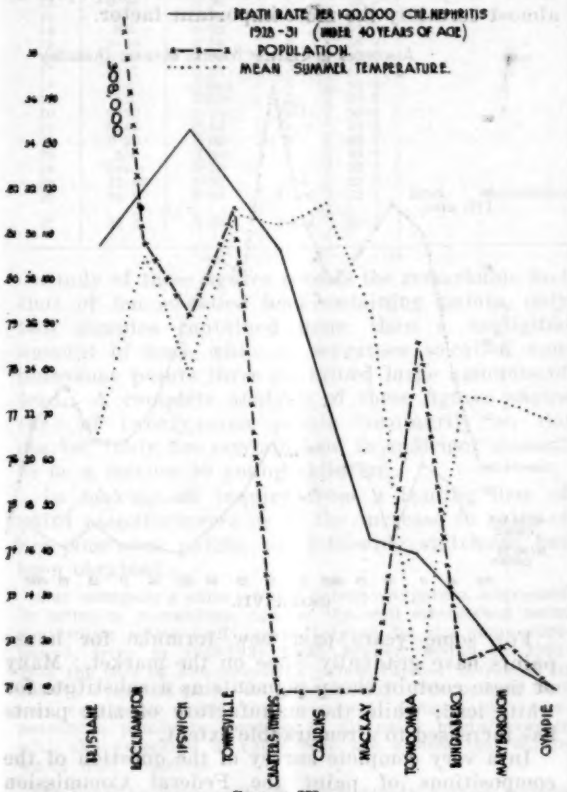
The mortality from nephritis in age groups in the main cities in Queensland has been collected by the Registrar-General for the past four years, and although this is too short a period in which to make definite deductions, careful study of these figures reveals some very interesting facts. It is noted that the greatest mortality from chronic nephritis among persons under forty years of age occurs in the cities of Rockhampton, Ipswich, Townsville and Brisbane.

In reviewing the incidence of chronic nephritis in the various cities of Queensland for the two years 1928 and 1929, the Federal report states:

> It appeared perfectly obvious, therefore, that in these particular cities a definite factor was causing a mortality more than four times as great as elsewhere for chronic nephritis before the age of forty years. It was just as apparent, however, that the same circumstances did not occur in other cities where conditions of life were apparently similar, but that, on the contrary, in such places very definite similarities, not only with the rest of Queensland, but with the rest of Australia were found, irrespective of the fact that they included every range of climate in Queensland.

A reasonable explanation of this distribution is given by a study of Graph VI, showing the mortality from nephritis among persons under forty years of age in the years 1928, 1929, 1930 and 1931, the population and mean summer temperature of each town.

It will be noted that Brisbane, Townsville, Rockhampton and Ipswich are the towns with the greatest mortality from nephritis and are also the towns with the largest industrial populations. This is additional evidence against lead paint, as it is obvious that in the larger cities children are more continuously kept on the verandas owing to the risk of injury from traffic if they were allowed to wander about freely.



GRAPH VI.

The case of Toowoomba is of interest, for, although it has a population of 25,000 (almost as many as Rockhampton, Townsville and Ipswich), it is noted that the deaths from chronic nephritis are considerably less than in these cities. When one considers that Toowoomba is two thousand feet above sea level, and its climate is very mild, it can be readily understood that children are not confined to the verandas in that city for long periods owing to the risk of catching a chill. A consideration of the mean summer temperature suggests that climatic factors have some bearing on the question, for it is observed that of the lesser cities, those with a high temperature have the greatest mortality from chronic nephritis; thus it is noted that in Charters Towers, Cairns and Mackay, all cities with high summer temperatures, the mortality is high. This would be expected, as in the very hot climates children are confined to the verandas, not so much on account of the traffic, as in the larger towns, but because the sun is too hot out of doors.

Although these figures are so small that one is hardly justified in drawing conclusions from them, nevertheless they lend support to the veranda railing hypothesis.

Nail-Biting.

As the habit of nail-biting forms such important evidence, it was considered necessary to make a special study of this question. It has been shown that in both Gibson's ocular neuritis series, in my nephritis series, and in the Federal series, the great majority of the patients were nail-biters.

As a proof that nail-biting is not such a common habit as to make the relationship coincidental, a control was obtained by the examination of one thousand consecutive State school children, and it was found that only 13·7% had developed that habit.

Paterson,[20] during his term of three years as Medical Superintendent of the Brisbane Children's Hospital, has observed only two cases of acute plumbism in children who were neither nail-biters nor thumb-suckers. They had both been living in a house where the paint was dry and powdery. It was his opinion that they had probably licked the paint dust from the railings.

Again, it is noted frequently that in one family it is only the nail-biters who become affected with nephritis, while the non-nail-biters remain healthy. The following is such a family history:

Isabelle, non-nail-biter, died goitre, age thirty-six.
Florence, nail-biter, died chronic nephritis, age twenty-two.
Albert, non-nail-biter, well, age forty-one.
Mildred, non-nail-biter, well, age thirty-six.
Margret, non-nail-biter, well, age thirty-four.
Ruby, nail-biter, advanced chronic nephritis, age thirty-two.
Audrey, non-nail-biter, well, age twenty-four.

The Federal report found:

In twenty-one cases the only member of the family who had the habit of nail-biting and thumb-sucking, or both, suffered from chronic nephritis, while the remaining members went free. In five cases two members who were nail-biters were found with the disease, while the remainder escaped.

The Federal report suggests:

The possibility that nail-biting may be a nervous reaction to irritation of the nervous centre produced during the circulation in the blood of lead, and therefore a sequel rather than a cause of plumbism, has not been sufficiently investigated.

A close study of nail-biting shows that it is an inherited familial habit in persons with nervous temperaments.

To confirm this, Bostock[21] examined a series of fifty consecutive nervous patients at the Brisbane Hospital; of these 38% gave a history of nail-biting, and 68% of these gave a definite history indicating a familial tendency to the habit. He stated that this was almost certainly an under-statement, since a number had little knowledge of the habits of the rest of the family.

In order to obtain further evidence, I questioned twenty-five nail-biters who could give accurate information concerning other members of their families. In twenty-one instances some other member or members of the family possessed the same habit.

This familial tendency is good evidence against a suggested *propter hoc* cause of the habit. Furthermore, McCann, in the Federal report, found

several families where all members were nail-biters, but only one or two had developed plumbism or nephritis.

The Federal report suggests a "possible familial factor" in chronic nephritis. This is well known to many practitioners in Brisbane, and it is my belief that the familial habit of nail-biting and familial susceptibilty to plumbism, as previously discussed, are responsible for the fact that one frequently sees a whole family affected with chronic nephritis.

The Federal report draws attention to the fact that:

No cases of lead poisoning with paralysis have been observed by the writers during the course of investigation.

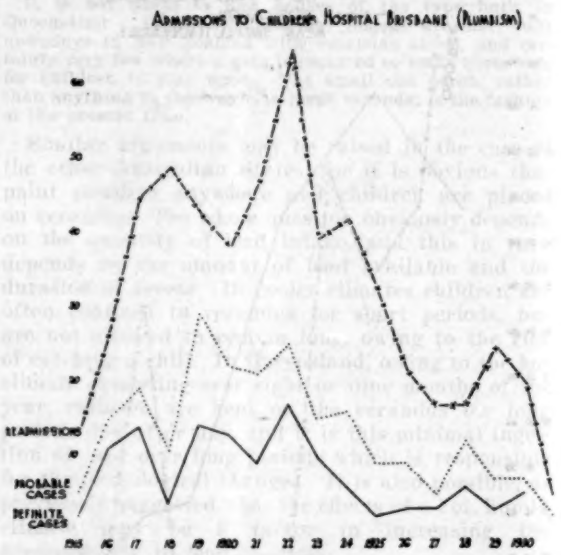
Although it is true that no patients with paralysis have been admitted to the Children's Hospital during that period, to my knowledge they have been observed by private practitioners. Dr. Earnshaw[22] and Dr. Harold Crawford[23] have both supplied me with the notes of definite cases with paralysis occurring during the past two years, while a considerable number of patients with the lesser signs are still being observed. In any case, in the past there were several years in which as few as two patients with paralysis were admitted to the Children's Hospital. It must be obvious that for every child who becomes paralysed by lead there must be literally hundreds who have been affected by the poison in some more or less minor degree.

We believe that the frequently accepted view that "without palsy there had been no lead" has had far-reaching and most unfortunate results. The whole issue has been clouded, the extent of the lead paint menace has been minimized, and in consequence, literally thousands of children have been allowed to run the risks of lead absorption.

It is certainly true that the cases of lead poisoning amongst children reported annually are gradually becoming less. The number of patients admitted as in-patients to the Hospital for Sick Children, Brisbane, diagnosed as suffering from plumbism since 1915 are as shown in Graph VII.

It is also interesting to note that the cases of severe chronic lead poisoning with marked foot-drop are now not frequently seen. This happier state of affairs is probably brought about by the operation of many new factors, such as the education of the public as to the dangers of lead poisoning from paint, legislation against the use of lead paint on veranda railings, the post-war period of prosperity, smaller families, earlier recognition and treatment by the medical profession, improved hygiene in the home and at school, the work of the Crèche and Kindergarten Society, a change in the type of houses, and finally the introduction of an enormous number of non-poisonous paints.

Although it may be debated that each of these factors does not contribute largely to the lessened incidence of lead poisoning, it is obvious that in the aggregate they constitute a very valid explanation of the gradual falling off in numbers and diminished severity of the cases now seen. Amongst these the introduction of non-poisonous paints is almost certainly the most important factor.



GRAPH VII.

For some years past new formulæ for house paints have gradually come on the market. Many of these contain cheap pigments as a substitute for white lead, while the manufacture of zinc paints has increased to a remarkable extent.

In a very complete survey of the question of the compositions of paint the Federal Commission has investigated twenty-seven brands of paint "ordinarily on the market". These results are shown in Tables II and III.

TABLE II (Federal report).

Showing Results of Analysis of Samples of Lead Containing Paint.

Sample.	Compounds of Lead Removed by—				Remarks.
	HCl 0·25% $PbCO_3$ and PbO_2.	Ammonium Acetate $PbSO_4$.	Nitric Acid Pb.	Vehicle Percentage.	
1	0·17	0·02	Nil	37·1	Effervescence with HCl.
2	Trace	0·01	Nil	29·8	
3	0·53	0·34	Nil	36·0	
4	0·28	0·02	Nil	50·5	
5	53·17	8·2	Trace	35·8	Marked effervescence with HCl.
6	Trace	0·04	Nil	27·2	Vehicle partly insoluble removed as film overlying pigment.
7	23·53	6·58	Trace	35·6	Effervescence with HCl.
8	Nil	Nil	Nil	51·2	
9	Trace	0·02	Nil	31·9	
10	Trace	0·03	Nil	39·0	

TABLE III.

Showing Results of Analysis of Samples of Non-Poisonous Paints.

Sample.	Compounds of Lead Removed by—				Remarks.
	HCl 0·25% $PbCO_3$ and PbO_2.	Ammonium Acetate $PbSO_4$.	Nitric Acid Pb.	Vehicle Percentage.	
11	0·85	0·25	Nil	40·2	
12	0·38	0·18	Nil	33·3	
13	Nil	Trace	Nil	35·3	
14	Nil	0·02	Nil	34·9	
15	0·45	0·13	Nil	38·9	
16	0·39	0·08	Nil	38·6	
17	0·47	0·21	Trace	43·9	
18	Nil	Trace	Nil	41·8	
19	Nil	Trace	Nil	40·9	
20	0·30	0·10	Trace	55·4	
21	0·44	0·15	Trace	34·3	
22	0·25	0·02	Nil	43·7	
23	Nil	Trace	Nil	44·2	
24	Trace	0·10	Nil	32·8	
25	51·82	1·82	Nil	22·9	
26	52·2	0·08	Trace	23·1	Brisk effervescence with HCl.
27	51·2	0·05	Trace	20·1	

Study of these figures reveals the remarkable fact that of ten so-called lead-containing paints, only two samples contained more than a negligible amount of lead, while of seventeen so-called non-poisonous paints three contained large amounts of lead. A complete analysis of these figures shows that of twenty-seven paints "ordinarily on the market" only five contain lead in sufficient amount to be a menace to young children.

In making an inquiry from a leading firm of paint manufacturers as to the increase in sales of non-poisonous paints, the following statement has been obtained:

Our company's sales of non-poisonous paints, expressed in terms of percentage, against the well established paint containing white lead (manufactured by the same company), were in 1929-1930 290% higher, whilst in 1931-1932 the sale of non-poisonous paints further increased to 408·5% over the paint containing lead. We believe that our experience with the increased demand for non-poisonous paints is common to all paint manufacturers with the exception of those who are almost married to white lead interests.

It may be argued that these ready mixed paints are not usually used by the trade, but inquiry from several contractors indicated that the tendency nowadays was to buy manufactured paint, either ready mixed or in bulk paste, such as "Tinc Titanium" paste.

It is probable that the introduction of the modern non-poisonous pigments and compounds has been the greatest factor in the falling off of cases of plumbism.

The Federal report mentions that lead poisoning "does not figure in hospital records other than Brisbane". This is probably due to the fact that unless a medical man is familiar with plumbism in childhood, it would hardly be expected that he would make a correct diagnosis even when accompanied by typical paralysis, for the disease is so rare elsewhere and the average student is taught little of the signs and symptoms of lead poisoning, it is quite excusable if he were to diagnose the condition as infantile paralysis. In this respect the Federal report states:

Plumbism in children is conspicuously unimportant in the literature, even the classical text books affording the briefest passing references. It is, therefore, not surprising that the condition escaped attention for years and much hesitation has been shown in admitting it to be common in Queensland.

That lead poisoning in childhood does occur elsewhere in Queensland is amply demonstrated by the report of Breinl and Young[24] who, in 1914, described sixteen cases in which the diagnosis had been proved by finding excess lead in the urine; also by the following statement from Dr. Gilbee Brown, Medical Superintendent, Maryborough Hospital:

I have seen a fair number of cases of lead poisoning and we are this much alive to the liability of cases turning up that we have installed a small H_2S generator for testing. At present I have under treatment a case of infantilism with chronic nephritis which started originally as lead poisoning in childhood derived from veranda railings and I have two cases (a brother and a sister), one Jacksonian and the other generalized epilepsy. These derived the lead from a house just outside Rockhampton.

The Federal report also states that:

A very typical case of lead poisoning was seen at Rockhampton, in a child aged four, by the Commonwealth Medical Officer in 1931, and occasional cases of lead poisoning have been seen recently in Townsville.

Finally, if one peruses the records of the admissions to the Hospital for Sick Children, Brisbane, it is found that a number of patients are admitted from time to time from country centres.

Our observations on saturnine nephritis in Queensland are very closely borne out by leading authorities on lead in other parts of the world.

Battaglia,[25] who has done a great deal of experimental work with lead, and has also made careful histological study of the kidneys in men who have died of saturnine nephritis, is very emphatic that the outstanding feature in these cases is the vascular sclerosis. His conclusions are that the wrinkled saturnine kidney is ascribed to vascular nephrosclerosis, and particularly to progressive arteriosclerotic nephrocirrhosis (Lohlein, Aschoff, Herxheimer). He and his co-workers do not consider the conception of the *"Kombinationsform"* of Volhard and Fahr to be acceptable in a general sense or in the special sense of saturnine kidney. He considers that the histogenesis of saturnine kidney is that of vascular nephrosclerosis, that is, it arises from primary alteration of the vessels and not, as some authors state, from primary epithelial lesions; these only accompany the vascular lesions later.

Our study of lead poisoning in Queensland entirely bears out these contentions.

Legge and Goadby,[26] writing in 1912, stated:

Unless it were established that granular nephritis was present in a lead worker before commencement of lead employment we think it would be useless to endeavour to prove that the condition was independent of lead, despite its comparative frequency as a cause of death apart from employment.

Badham and Taylor[27] state:

To postulate that nephritis or arteriosclerosis will only follow in those lead workers who give a history of attacks of colic or palsy is at best only an intelligent guess and

in keeping with the fact that a history of typical colic or palsy proves that these individuals have been engaged in lead work. Such a position is tantamount to demanding a history of *delirium tremens* in a chronic drinker before relating an arteriosclerosis or nephritis to his liquor.

Legge,[28] while entirely accepting the veranda railing hypothesis, points out that it is known that the daily absorption of a grain may produce lead poisoning in an adult, and he remarks how infinitesimally small a dose would be required in order to produce similar results in a child of two or three years of age.

Oliver[29] states:

> Any effect produced by lead is not so much due to the amount of poison circulating in the blood at a particular time nor to its deposition in the tissues, but, as Straub suggested, to the summation of the injuries inflicted by its recurrent presence in the blood in infinitesimal quantities.

Aub[30] has shown that large quantities of lead are stored in the bones and are liberated only by some disturbance of the body metabolism; thus in one human skeleton he found as much as 800 milligrammes. In this fact probably lies the explanation of the slow and insidious progression of the renal and vascular sclerosis, till a stage is reached in which impairment of the function of the organ has occurred to an extent inconsistent with life.

It is well known that some chemical poisons have definitely selective actions on certain tissues, and it appears that the only satisfactory way of explaining the histological changes is by assuming that lead has some selectivity for the vascular endothelium, as maintained by Battaglia, for how else can one explain the generalized arteriosclerosis and the histological picture of our cases showing such marked diffuse vascular sclerosis.

Conclusion.

Fishberg[31] states that preeminent among the exogenous poisons which cause chronic hypertension in man, if indeed there are any others, is lead. When the clinical signs of a slow vascular sclerosis are so manifest, and when such a well recognized sclerosing agent as lead has been shown to be so commonly present in the childhood environment of these young people, it is difficult to understand why the true ætiology of many of these cases has been accepted so reluctantly.

After a careful review of the statistical, clinical, environmental and experimental evidence which has been collected in the Federal report and in this work, it must be admitted that lead poisoning in childhood is at least one factor which is responsible for the increased mortality from chronic nephritis in Queensland. Furthermore, the amount of positive evidence incriminating lead paint is so overwhelming that the introduction of further hypotheses before demanding preventive legislation would savour rather of the nature of a deliberate digression than of a search for clinical truth, for it must be admitted that the case against lead paint has been reasonably proven.

The purpose of this work has been twofold. First, it has a special application to the people of Queensland and forms the basis for an appeal for the introduction and enforcement of legislation against lead paint, thereby saving the lives of a great number of our young population. Secondly, it applies to every country in the world, since it teaches the lesson of the damaging effects of lead on young tissue, and should form the basis for a demand that lead in any form should be banished forever from those places which serve as a playground for restless and meddlesome infancy.

Acknowledgements.

I beg to acknowledge my indebtedness to the following: Mr. Geo. Porter, Registrar-General for Queensland, for supplying numerous statistics and tables, and for his ever-willing assistance in the dissection of such figures; J. B. McLean, General Medical Superintendent, Brisbane and South Coast Hospitals Board, for making available the records of the Brisbane General Hospital; A. E. Paterson, Medical Superintendent, Hospital for Sick Children, Brisbane, for making available and assisting in the dissection of the records of patients admitted to the Hospital for Sick Children, Brisbane; W. E. Bush, Chief Engineer, Water Supply and Sewerage Department, Brisbane City Council, for information concerning Brisbane's water supply; W. J. Chamberlain, Chief Chemist, Water Supply and Sewerage Department, Brisbane City Council, for special analyses of various waters and information on plumbisolvent qualities of waters; J. Butcher, Government Statistician, Dominion of New Zealand, for supplying New Zealand statistics and information as to types of houses; J. W. Davidson, Commissioner of Railways for Queensland, for data concerning the powdering of paint in districts throughout the State; to many medical practitioners, especially to Dr. John Bostock, who have referred many patients to me and have assisted in various ways in this investigation.

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THE TREATMENT OF UTERINE CANCER.

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IN *The British Medical Journal* dated November 19, 1932, there is published a paper by Dr. Victor Bonney on the treatment of uterine carcinoma by Wertheim's method of operation. In his summary of the operative mortality rate per hundred cases Dr. Bonney states that his latest results show a 9% post-operative death rate. The point I wish to raise is: Why persist in using Wertheim's method which is so obviously faulty from an anatomical and pathological point of view?

The whole operative procedure is intraabdominal, thereby rendering the last stages of the operation most unnecessarily difficult, and also positively asking for septic infection of the peritoneal cavity by contamination from the diseased and almost certainly septic *cervix uteri*.

As bearing upon this, I remember in 1914, shortly before the war, watching Dr. Bonney deal with a case of *carcinoma uteri* by Wertheim's method at the Middlesex Hospital. I noticed that at the end of the operation he used a vagino-abdominal drain. On asking him why he did this, his reply was that he feared post-operative sepsis if he did not do so. My reply was that during the previous ten years I had used a vaginal drain in only two cases, and that there had been no deaths from post-operative sepsis. But I did not follow Wertheim's technique.

The vagino-abdominal method, if efficiently carried out, completely avoids septic infection of the peritoneal cavity at the time of operation.

Briefly, the details are as follows. Before opening the abdomen, and with the patient in the "lithotomy" position, a thorough removal of the obvious cervical growth, together with any extension of the growth on to the vaginal wall, by means of a Volkmann's sharp spoon or a large, sharp, ringed curette, is done. (In passing, may I say that the so-called "blunt curette" is a most inefficient and dangerous instrument and should never be used.)

The surface left after the sharp-spooning is thoroughly swabbed with a solution of biniodide of mercury in spirit, one in five hundred, or other efficient antiseptic. Next, a circular incision is made through the vaginal wall, well below any extension of new growth which may have invaded the vaginal tissues, the upper section of the vagina is dissected upwards, and as soon as a sufficient cuff is made, the cut edge of the upper section of the vagina is tightly closed by a continuous suture, the *cervix uteri* and upper portion of the vagina being thus shut off and contamination of the peritoneal cavity during the later stages of the operation being prevented. At the same time the operator is enabled to free and lift the base of the bladder, trigone and lower ends of the ureters from the uterus in front, and also to free the peritoneum posterior to the uterus, and if it appears to be advisable, to open into the pouch of Douglas. This procedure very definitely makes the later intraabdominal portion of the operation easier and adds greatly to the safety of the lower ends of the ureters.

Next, with the patient in the Trendelenburg position, the intraabdominal portion of the operation can be made comparatively bloodless if the operator really knows his pelvic anatomy.

The uterus with its appendages, the whole broad ligament with its contained pampiniform plexus, lymphatic vessels and small lymphatic glands should be removed *en masse* and not in sections. This can be done with very small loss of blood.

Each round ligament is double-ligatured and divided; an incision is made through the peritoneum from one cut round ligament to the other; and the bladder is separated from the uterus completely. Next, the ovarian vessels in each infundibulo-pelvic fold are exposed and divided between two number 1 catgut ligatures, an aneurysm needle being used, no clamp. Divide the peritoneum between the cut end of each round ligament and the site of ligature of the ovarian artery on each side, the line of incision through the peritoneum passing along the side of the pelvis and below the brim. The peritoneum forming the broad ligament is then dissected away from the side of the pelvis and turned inwards, care being taken to detach the ureter from its posterior part. The nutrient vessels of the ureter can be seen and need not be harmed.

Next, expose the uterine artery far back near its origin from the anterior division of the internal iliac artery, using an aneurysm needle, not a clamp; tie with double ligatures and divide between. If the uterine artery is difficult to find, tie the anterior division of the internal iliac artery and divide between two ligatures. The collateral circulation is good. I have on three or four occasions divided the internal iliac artery between two ligatures, but this can seldom be necessary. The broad ligament lymphatic vessels can be followed up to

and into the pelvic lymphatic glands, which themselves should be carefully dissected away from the internal iliac vessels, a clean dissection of each side of the pelvis being left without damage to either vessels or nerves.

The lower ends of the ureters can be clearly exposed as they pass in the short tunnel deep to the now bloodless uterine arteries to enter the trigone of the bladder.

If there is much subperitoneal fat, the safety of the pelvic portion of the ureter can be assured by tracing it from the pelvic brim downwards and forwards and gently detaching it from the peritoneum which covers the side of the pelvis and the pelvic vessels and nerves. What is sometimes tiresome is the control of the vein or veins which return the blood from the pampiniform plexus to the internal iliac veins.

If there is only one large vein directed outwards and a little forwards, it is comparatively easy, by the use of an aneurysm needle and two number 1 or number 2 catgut ligatures, to divide the vein between the ligatures. Sometimes, instead of one big vein, there are several smaller veins, each of which should be dealt with in the same way.

I feel very strongly that the widespread and, may I say, reckless use of clamps within the abdomen, which has become customary of late years, is productive of a vast amount of unnecessary trauma. This throws a very great and uncalled-for strain upon the recuperative powers of the peritoneum, due to the presence of crushed and devitalized tissue which has to be dealt with by the peritoneum.

This leads me to the last stage of the operation, namely, the closure from within the abdomen of the lower segment of the vagina by means of a continuous catgut suture.

If the arteries and veins have been dealt with as I have described, very little blood will have been lost and the pelvic cavity will be practically "dry" but for serous oozing.

For the last thirty years I have been enabled to reduce post-operative drainage of the abdomen in abdominal operations of all kinds to a minimum.

To do this unnecessary trauma must be avoided and the phagocytosis of the peritoneum stimulated by filling the abdominal cavity with all the normal saline solution which it will hold, the patient being in the Trendelenburg position. This procedure not only stimulates the phagocytosis, but gives the patient the fluid so much needed after a somewhat severe operation and floats the various abdominal contents to normal positions.

By following the practice which I have, perhaps somewhat sketchily, described, I find that my post-operative mortality rate was, some twenty years ago, reduced to 3·5% to 4%. It certainly has not increased since then.

There was no picking or choosing for statistical purposes, but all "border-line" cases were operated upon.

As an instance, one patient at the Royal Prince Alfred Hospital, Sydney, New South Wales, whom I considered definitely a "border-line" case, I examined before handing my ward over to the assistant surgeon for the usual four weeks in the year. An operation was commenced by him, but was abandoned as "inoperable".

On my return to take over again, I found the patient still in the ward and still, in my opinion, a "border-line" case.

The operation above described was performed, the patient recovered, and fourteen years afterwards was again admitted under me at the Coast Hospital, New South Wales. The then diagnosis was a probable duodenal ulcer. I operated, doing a gastro-jejunostomy, "no loop". The patient is, I understand, still alive and well. At the second operation I took the opportunity of examining as far as was possible the first operation area. There were no adhesions; all was clear in the pelvis.

I may add that in almost all cases of the operation for *carcinoma uteri* I removed the *appendix vermiformis*.

Next, as to the value of statistics, either the massed statistics of many operators or the statistics of individual operators. The real value of statistics must be checked by a knowledge of the individual operator. Is he (or she) merely a skilled "surgical mechanic" who blindly follows "So-and-So's" method as set forth in a text book, or is he gifted with the virtue (?) of individuality and capable of grasping the fact that real surgery is as described by the late Dr. Erichsen in his "Manual on Surgery", a "science and art".

The question may be asked: why have I written all this and gone into these details even sketchily?

A partial answer is that, while I fully appreciate the immense utility of the recent advances in the application of X rays and the use of radium, each one of these aids to surgery has been abused and misused. This especially applies, I believe, to the application of radium emanations to deep-seated malignant growths, such as secondarily infected pelvic glands, in cases of *carcinoma uteri*.

What is the effect of radium emanations which, in order to reach an extension of or secondary deposit of carcinoma, say, in the deep pelvic glands, have to pass through normal healthy tissues and organs, such as blood vessels, nerves, ureters, before playing upon the carcinoma-infected glands themselves? (Eric W. Frecker, THE MEDICAL JOURNAL OF AUSTRALIA, February 4, 1933.)

While freely admitting the good results which are being obtained upon superficial malignant growths, I still believe that well-directed and executed surgery holds first place in the treatment of deep-seated primary growths and secondarily infected lymphatic glands.

Here may I make a protest against the loose way in which the word "recurrence" is used in connexion with cancerous or malignant growths? In practically all cases the so-called "recurrence" is simply the development of a focus dependent upon the primary growth and existing, but not dealt with and removed, at the time of the original operation.

I may add that in still advocating the vagino-abdominal operation for *carcinoma uteri*, my first case was done in December 1895. It was compli-

cated by double ovarian cysts and pelvic adhesions. I was privileged in having Dr. (as he then was) Charles J. Martin, the then lecturer in physiology at the Medical School in Sydney, New South Wales, to assist me. He also reported upon the specimen removed as being squamous epithelioma of the *cervix uteri*, together with secondary carcinomatous deposit in two of the deep pelvic glands on the left side. This patient is still alive and well.

One other fact is, I think, worth recording. The post-operative mortality rate for my first twenty-seven cases was 12%; the next twenty-five cases gave no post-operative deaths. This decided me that the operation was justifiable. It was interesting, too, that before operating upon my first patient, referred to just above, I consulted Professor J. T. Wilson, then occupying the Chair of Anatomy at the University of Sydney and now at Cambridge, as to the feasibility of carrying out the operation. His comment was that, while theoretically correct, it could not be done on the living body.

This was not encouraging, but I ventured to differ.

As a justification for a minimum use of so-called drainage of the peritoneal cavity after abdominal operations of all kinds, I would refer those who are interested to the Cavendish Lecture on "Experiments on the Immunisation against Infection, of Operation Wounds, Especially of the Peritoneum", by Johann von Mikuliez-Radecki, M.D., LL.D., published in the issue of *The Lancet* of July 2, 1904.

Since that date I have in almost all cases of abdominal surgery used normal saline solution to fill the peritoneal cavity, the patient being in the Trendelenburg position, and then closed the abdominal cavity without drainage of any kind. This procedure was made use of in operations upon the liver, stomach, small and large intestine, spleen, and pelvic contents.

In order to illustrate I will describe shortly three cases.

T.J.W., a boy, aged four years, had an illness of three to four days' duration. He was seen by Dr. E. W. Fairfax at Camden, New South Wales. Dr. Fairfax diagnosed intussusception of the colon, with which diagnosis I agreed. He brought the boy to Sydney and the same evening we met at "The Terraces" Hospital and decided to operate at once. An incision was made in the left iliac region through the outer side of the left rectus sheath. On opening the peritoneum thick pus poured out; no intussusception was found. Next, an incision was made in the right iliac region, the caecum was exposed, together with the largest and longest appendix I have ever seen. Pus apparently filled the whole abdominal cavity. The appendix was removed and was subsequently found to be full of pus.

A third incision was made through the inner side of the left rectus sheath, above the umbilicus, and the whole abdominal cavity was thoroughly irrigated with normal saline solution until the fluid returned clear.

The three abdominal incisions were closed without drainage. Convalescence was uneventful. I last saw the patient a year ago, aged twenty-seven years, well and "fit".

Mrs. C., aged forty years, gave a history of repeated and severe attacks of "pelvic peritonitis". The diagnosis was double pyosalpinx. The abdomen was opened. Each Fallopian tube was full of pus, and with its ovary densely adherent to the posterior surface of the broad ligament and pelvic floor. Both tubes and ovaries were dug out and removed. The top of the broad ligament was closed with a continuous suture of fine gut. The abdomen was filled with normal saline solution. The abdomen was closed without drainage.

Twelve years later the same patient consulted me again. Examination showed a solid mass the size of a grapefruit, occupying the position of *caput caecum coli*. The diagnosis was probably carcinoma of the gut.

At operation it was seen that the *appendix vermiformis* had been removed at the first operation. An incision into the *caput caecum coli* revealed a large stereolith, the walls of the gut being greatly thickened. The hard fæcal mass was removed, and the gut was sutured. An examination of the pelvis and previous operation area showed a fine white cicatrical line along the top of the broad ligament on each side of the uterus, and no pelvic adhesions. The abdomen was again filled with normal saline solution and closed without drainage. The patient was alive and well when last seen by me, six years after the second operation.

A third case is worth recording as bearing upon the drainage question.

Mrs. M., aged thirty-two years, for eighteen months before being admitted to the Royal Prince Alfred Hospital under me, complained of varying degrees of discomfort in the lower part of the abdomen and of irregular action of the bowels. For the two weeks previous to admission to hospital the lower abdominal pain had been severe, accompanied by irregular rises of temperature, up to 38·9° C. (102° F.). Examination disclosed a solid or semi-solid tumour occupying the right side of the pelvis and rising well above the pelvic brim, fixed firmly to the side and floor of the pelvis. The *appendix vermiformis* could not be found until, by prolonged and careful dissection, it was found attached to the tumour and the side of the pelvis, over the iliac vessels and ureter. The mass proved to be an immensely thickened *caput caecum* with the *appendix vermiformis*, which latter contained a large concretion just about the size and shape of one's thumb, including the metacarpo-phalangeal joint.

While freeing the mass a Y-shaped slough on the inner and lower portion partly separated and from the hole exuded fæcal material.

The ascending colon was divided distal to the mass, the cut end was closed, the ileum was divided some inches from the mass, cut and closed. The tumour was removed and a lateral anastomosis was made between the ileum and ascending colon.

The operation was difficult and the patient's condition showed the effects of the prolonged manipulation. I decided not to drain either through Douglas's pouch or the abdominal incision, but to fill the abdomen with saline solution and close it. At the end of a week the patient's temperature was normal. On vaginal examination there was a certain amount of "thickening" in Douglas's pouch. There were no signs of pus, and at the end of three weeks the patient was out of bed and, so far as I have been able to ascertain, has remained well since.

The reader is probably asking himself what have these three cases to do with uterine carcinoma.

I have simply quoted them in order to demonstrate that if you treat the peritoneum as a friend, assist its production of phagocytosis by the free use of normal saline solution and lessen the load it has to carry by avoiding unnecessary trauma, you can reduce the post-operative mortality rate, not only in doing a radical operation for *carcinoma uteri*, but in all kinds of abdominal operations.

The post-operative mortality rate in *carcinoma uteri* should not exceed 3%. I therefore still believe that for the present at all events surgery will give better final results than radium.

In The Medical Journal of Australia, February 4, 1933, I read a paper by Eric W. Frecker,

headed "A Working Hypothesis of the X Ray Effect". While fully and freely admitting my profound ignorance as to X rays and their effect upon living tissues, I could not help a feeling that there is still a good deal of assumption, supposition and theory, not sufficiently supported by facts, in connexion with X ray treatment of deeply placed malignant growths.

To obtain a minimum post-operative mortality and morbidity rate three things are essential: (*a*) effective control of blood vessels; (*b*) gentle handling of tissues, and especially a minimum use of clamps; (*c*) the full use of the phagocytic action of the peritoneal endothelium, stimulated by the use of normal saline solution filling the closed peritoneal cavity. This also acts, I believe, by washing away or diluting the lymph effused from points or areas within the peritoneal cavity where unavoidable damage has been inflicted during the operation. In this way it is conceivable that the usual, or at all events frequent post-operative adhesions can be avoided or reduced to a minimum.

I should like to add that, with a few exceptions, I did not use rubber gloves myself; the assistants and nurses did in all cases since the introduction of the custom.

Lastly, may I express the very definite opinion that modern surgery is suffering from the evil of "over mechanization", as is the world in general.

There is a pernicious attempt being made to limit and define certain "specialties" and to attempt to make the patient fit the method, instead of fitting in each case the method to the patient.

Appendix.

Since writing my paper it occurred to me to look up the notes of the following case. They illustrate the effects of deep X rays to the surgical treatment of carcinoma of the uterus in a way which is unlikely to occur often.

Mrs. T., aged thirty years (?), was operated upon by me for double pyosalpinx on November 22, 1911. Both Fallopian tubes and the ovaries, which were hopelessly involved in adhesions, were removed, and also the *appendix vermiformis*. She made a complete and uneventful recovery.

In August, 1921, ten years later, she was operated upon by Dr. F. Barrington for *carcinoma uteri*. He opened the abdomen, intending, I believe, to follow Wertheim's method, but closed it again without removing the uterus, regarding the case as "inoperable". For some months after this operation the patient was treated with deep X rays, I think by Dr. Molesworth. She came to me again in October, 1923 (that is, two years), being then in a very miserable state. She was thin and emaciated, there was a profuse and fœtid purulent discharge from the vagina; she had constant and severe pain in the left hip and shooting down the sciatic nerve.

On examination I found the uterus *in situ*, and there was a fixed mass in the left posterior quarter of the pelvis.

In order to give relief, if possible, from the misery she was suffering, but without holding out hope of permanent cure, I decided to do the vagino-abdominal operation.

The vaginal part of the operation was done in the usual way. The abdominal scar was excised. Dense and extensive adhesions of gut and omentum were separated. The uterus and broad ligament were dealt with in the way previously described, and the mass of infected glands in the left posterior quarter of the pelvis was dissected away from the vessels and nerves.

The patient made an uneventful recovery. She lived about two years in relative comfort. No more of the fœtid vaginal discharge, and no more of the intense sciatic pain due to pressure by the enlarged glands.

The specimen was sent to Dr. Keith Inglis for a report, which is as follows:

"Craignish",
185, Macquarie Street,
30/10/23.

Mrs. T. Specimen Uterus and Lymph Gland.

Dear Dr. Thring,

These specimens have been examined with the following results.

Macroscopic.

(*A*) The uterus was ulcerated and excavated in lower portion, but section through the uterus wall did not show opaque areas of infiltrating neoplasm.

(*B*) A small piece of tissue apparently including external os.

(*C*) Large lymph node with much necrosis.

Microscopic.

(*A*) The ulcerated area is composed almost entirely of granulation tissue; very little epithelium is to be seen, but a few deeply placed masses of squamous epithelium can be made out.

(*B*) This shows cervical mucous glands and much inflammation; but no neoplastic tissue was detected.

(*C*) The gland is extremely infiltrated with squamous epithelioma which is forming the walls of large cystic spaces containing necrotic growth.

Though the growth shows definite metastatic spread the histological picture is in keeping with the clinical history (two and a half years' duration) and suggests that the malignancy is not quite as great as is usually the case with this type of neoplasm.

Yours sincerely,
KEITH INGLIS.

It is of great interest because of the fact that the material examined was removed from a living patient after prolonged and intensive treatment by X rays. The case has, I think, a definite bearing upon the relative value of initial treatment of cancer of the uterus by surgery or by deep X rays.

Finally, may I plead for a broader view with regard to so-called gynæcology?

I did this in 1908, when, during the Medical Congress meeting for Australasia, as President of the Section of Obstetrics and Gynæcology, I definitely stated that no man should practise as a gynæcologist unless he was prepared to deal with any intraabdominal condition, or, in other words, that what is called gynæcology should be regarded as belonging to abdominal surgery as a whole. The pioneers of abdominal surgery were obstetricians and gynæcologists.

It would help greatly in reducing the percentage of post-operative mortality and morbidity rates if the following simple facts were more clearly and definitely realized and put into practice. Look upon the peritoneum as a friend and not an enemy to be feared.

Any damage to tissues within the peritoneal cavity means an effusion of lymph and subsequent adhesions of gut, omentum *et cetera*, unless definite and adequate measures are taken to prevent the formation of adhesions.

The abuse and over-frequent use of drainage tubes is a potent cause of post-operative adhesions.

The use of peritoneal drainage, so-called, by any method can and should be reduced to a minimum if efficient means are used to promote phagocytosis.

In the matter of sutures and ligatures, I never use silk, but always carefully prepared catgut, never thicker or heavier than number 2, which becomes absorbed after its work is done, not encysted, as silk frequently is, thus remaining as a very possible source of trouble later on.

One would almost be justified in writing an article on "the use and abuse of sponges or pads within the abdomen", under headings such as "how to place sponges or pads in position for various operations" and, above all,

"how not to use a dry gauze sponge to arrest oozing or wipe away blood, but in effect to damage widely the delicate peritoneal endothelium by unnecessary friction".

Much of the present day ritual in abdominal surgery resembles very closely the pharisaical methods spoken of in Scripture, that is, the washing of hands and of the outside of the cup and platter, but leaving the inside to take care of itself.

The Cavendish Lecture, to which I referred before, is well worth the attention of the present day abdominal operator.

THE TREATMENT BY RADON OF THE GLANDULAR EXTENSION FROM A MALIGNANT CERVIX.[1]

By L. S. KIDD, M.D. (Melbourne), F.R.A.C.S.,
Melbourne.

THE subject of this small paper is the treatment by radon of the glandular extension from a malignant cervix.

It is generally accepted that once a carcinoma of the cervix has healed after efficient treatment by radium it should not reappear locally. The subsequent failures of some initial or apparent successes are due to invasion of the glands. There is a marked fall of the percentage rate of cure as soon as the invasion has spread to the parametrium. In order to get radium within effective range of this area, several plans have been evolved; some are still on trial, others have been abandoned. I beg to lay before you a technique I have been using during the past year.

Eight weeks after the vaginal application of radium to the cervix has been made, and provided the response has been satisfactory, the abdomen is opened and a careful inspection of the pelvis is made. The ovarian vessels and the round ligaments are separately ligated, traction is put on these ligatures so that the broad ligaments can be opened right down to the cervix, and the peritoneum is lifted off the external iliac vessels. A good view of the whole parametrium is obtained and the site is ready for the radon.

Thirty-two seeds are used in the following way. Four pieces of number 6 gauge soft rubber tubing, each about 25 centimetres long, are required. Into one end of each tube a series of eight radon seeds is inserted and spaced at regular intervals of one centimetre. These seeds are kept in place by a silk ligature tied firmly around the tube, a small compartment being thereby made for each seed. We thus have ready four rubber tubes 25 centimetres long and each charged over the terminal eight centimetres with eight radon seeds.

A tube is laid in the bottom of the split broad ligament, the end being close to the cervix, lying parallel to the uterine artery, then up along the inner pelvic wall, over the external iliac artery, and through a tunnel under the peritoneum of the iliac fossa and out through a stab wound near the anterior superior spine, the radiating section of this tube lying between the cervix and the pelvic brim.

[1] Read at the Fourth Australian Cancer Conference, Canberra, March, 1933.

Another tube, similarly loaded, is placed just below the external iliac artery, its inner tip near the sacro-iliac joint and extending to the obturator foramen, then upwards behind the peritoneum and out through a stab wound near the internal inguinal ring. The radiating section of this tube extends from the sacro-iliac joint to the obturator foramen. The same procedure is repeated on the opposite side. In five to seven days the tubes are easily withdrawn.

The dosage from the 32 radon seeds is 48 millicuries, equivalent to about 6,000 milligramme-hours.

Partly owing to the lightness of the screenage, the reaction in my cases was marked, the temperature rising to 38·3° or 38·9° C. (101° or 102° F.), but the pulse rate kept about 100. There was a moderate degree of ileus and tenderness was present over the lower part of the abdomen, but the bowels acted without any trouble. There was no vomiting. The induration in the parametrium increases for a fortnight, the uterus becomes fixed, and this pronounced thickening takes about ten weeks to subside, and in six months the uterus is moderately mobile, but it has atrophied considerably.

I have had practically no bladder irritation, but there has been a fair amount of rectal tenesmus lasting about fourteen to twenty-one days and starting on about the tenth day; it was, however, relieved by starch and opium enemata.

After a period of six months an opinion concerning the advisability of deep X ray therapy is sought. The object of this paper is to stress the necessity for an attack on the glands and to suggest that the above technique with radon might possibly prove to be of value in selected cases; but it is one step only in the plan of campaign.

Reports of Cases.

A CASE OF INFANTILE PARALYSIS: THE PREPARALYTIC STAGE WITH RECOVERY.

By H. A. MAUNDER, D.S.O., M.B., B.S.,
Warracknabeal, Victoria.

Clinical History.

ON Thursday, May 19, J.B., a female, aged seven years, was feverish and listless, had vomited and complained of a slight pain in the neck. She was seen by me at 8 p.m. on Thursday. Her temperature was 38·9° C. (102° F.), her pulse rate was 114, and her respirations numbered 36 in the minute. Her neck was tender, but not markedly so, on the right side, behind the sterno-mastoid muscle. There was no neck rigidity nor sore throat. The central nervous system signs were normal. I suspected an attack of influenza, and as the child had had lobar pneumonia twelve months previously, I paid particular attention to the chest.

On Friday, May 20, the child was much better. The temperature was 37·2° C. (99° F.), the pulse rate was 90, and the respirations were 22. The neck was not so painful, and no other signs had developed. At 8 a.m. on the Saturday (the day following) I was called to the child and found her much worse. Her temperature was 37·8° C. (100° F.), her pulse rate was 120, and respirations were 30. The pain in the neck was now severe, the child was very irritable and could not bear to be touched; she was

also very tender in the lumbar region. There was some neck rigidity, and Kernig's sign was present, I thought (this was difficult to say with certainty, as the child was so irritable). The reflexes were all normal.

At this stage I suspected the preparalytic stage of infantile paralysis and communicated by telephone with Dr. R. Southby, medical officer to the committee controlling the poliomyelitis campaign. He agreed with this provisional diagnosis and with the utmost expedition he arranged to despatch by the Royal Australian Air Force 70 cubic centimetres of poliomyelitis serum. This arrived at 3 p.m. and was dropped in the main street of the town. A lumbar puncture was done under a general anæsthetic; the fluid was under very slight pressure and was clear. Examination under the microscope revealed an average of seven to nine cells per field. Fifty cubic centimetres of serum were then injected—ten cubic centimetres intrathecally, twenty cubic centimetres intravenously, and twenty cubic centimetres intramuscularly. The child at this stage looked desperately ill.

The next morning the child was much better; the neck stiffness was not so marked, she was not irritable, and the tenderness of the back was not present. Her knee jerks, however, were absent and the left superficial abdominal reflex was weaker than the right. These signs persisted for about a fortnight. Beyond this the child made an uneventful recovery.

Comment.

This case is of interest in showing the wonderful response to treatment. On the Saturday afternoon the child was extremely ill; twenty-four to thirty-six hours after the injection of the serum she was well on the way to recovery. It would appear that delay in this case would have resulted in the onset of paralysis, and owing to the expedition with which the serum was dispatched, a tribute must be paid to the medical officer to the committee controlling the poliomyelitis campaign, Dr. R. Southby, and to the Royal Australian Air Force.

Reviews.

THE TROPICAL DOMICILE.

The living conditions of the natives of tropical countries form the subject of a small book by D. B. Blacklock, bearing the dignified title, "An Empire Problem".[1] Dr. Blacklock is Professor of Parasitology in the Liverpool School of Tropical Medicine, and is able to speak with authority. Objects, among others, of the book are to draw the attention of non-medical readers to "certain problems, upon the solution of which the whole future of the natives of our tropical possessions depends", and to suggest that the cooperation of governments and large industrial enterprises is necessary for true progress in sanitation in the tropics. He points out that the application of measures of disease-prevention on a large scale is beyond the power of limited medical and health staffs in tropical countries.

It is difficult to make people see how the living conditions (hence the health) of natives in remote hot climates can possibly be of importance to the British Empire as a whole, and still more difficult to persuade them to throw some of their energy and resources into the task of improving those conditions. Professor Blacklock employs convincing arguments that depend largely on his discussion of the disease, parasites and pests that are able to attack, batten upon and annoy the native in the wretched conditions of sanitation and hygiene under which he is forced to exist. He points out how all these things may be defeated. The improvements he suggests are desirable; but he sets perhaps too high an ideal at this stage, and for this reason his campaign may fail. For instance, he would provide the native with a staunchly built house with a smooth floor of concrete or other suitable, hard, impervious substance, a framework of metal and a roof of iron coated on its under surface with some non-conducting substance. The suggestion comes as a shock to anyone who has been in the tropics and has seen white men quite happy with their lot in houses with thatched roofs and bamboo walls. Professor Blacklock points out ways by which it might be possible to provide native living conditions approaching the ideal. They are not impracticable, or, rather, they would not be impracticable if governments and industrialists would realize the necessity for them.

This little book is commended to everyone. Whether it achieves its object or not, it will certainly provide interesting reading for all who care to spend an hour over it.

[1] "An Empire Problem: The House and Village in the Tropics", by D. B. Blacklock, M.D.; 1932. London: Hodder and Stoughton, Limited. Crown 8vo., pp. 100. Price: 3s. 6d. net.

HINTS ON HYPNOSIS.

Alexander Cannon's tiny volume on "Hypnotism, Suggestion and Faith-Healing"[1] is far from satisfactory either for a student or the general practitioner of medicine. It is too fragmentary and sketchy in style. It purports to set out the methods of Lloyd-Tuckey, Bernheim, Grossmann, Liebeault and others, but those portions of the book which are thus devoted to these special methods are more like the hasty notes of some medical student taken at a lecture than the words of the lecturer himself.

The book is lamentably uncritical. It suggests half a dozen or so different methods of inducing hypnosis without attempting to show which methods might be used to the best advantage according to the type of patient or the neurotic state one may be dealing with.

The author makes extravagant claims as to the value of hypnosis in nearly all forms of insanity, with the exception of *dementia præcox* and paranoia. While the larger portion of the book is devoted to the means of putting the patient into a hypnotic and receptive state, no attention whatever is paid to the therapeutic suggestions which must then be given. The author certainly recognizes the value of hypnosis in medical practice, but there will be few who read his pages, we think, who will agree with his claim that "this work contains all that one need know".

[1] "Hypnotism", by Alexander Cannon, M.D.; 1932. London: William Heinemann (Medical Books) Limited. Crown 8vo., pp. 60. Price: 2s. 6d. net.

PHARMACOPŒIAL DOSES.

Byron L. Stanton has compiled a small book dealing with the doses of preparations in the 1932 pharmacopœia and related matters.[2] The official doses of the drugs and preparations are alphabetically tabulated. Then follow classified doses of the acids, alkaloids, aromatic waters, extracts, infusions, liquors *et cetera*. A section deals with standardized preparations of drugs and biological products, such as cinchona, digitalis, *nux vomica*, opium, antitoxins, irradiated ergosterol, and so forth. The strengths of the more important preparations are given. This little work should be useful for reference, but is too large to be carried easily in the pocket. The pharmacopœia urges the use of Arabic, not Roman numerals, but in the footnote on page 22 we see a dose expressed in Roman figures. Where the new doses differ markedly from those of the previous pharmacopœia, as in the cases of tinctures of *nux vomica* and belladonna and the single doses of digitalis preparations, it would be well to draw attention to the differences. The drastic change in the dose of dry extract of hyoscyamus might have been indicated.

[2] "Classified Doses of the British Pharmacopœia, 1932", compiled by B. L. Stanton, M.R.C.P., M.B., B.S., F.P.S.; 1933. Melbourne: W. Ramsay. Crown 8vo., pp. 45. Price: 3s. 6d. net.

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ARTHRITIS.

During the last few decades much progress has been made in certain branches of medicine. Medical practitioners can look with satisfaction on the newer knowledge of the anæmias, on the successful treatment of general paralysis of the insane by the induction of malaria, on the advances made in brain surgery, and on many other additions to knowledge and to the therapeutic equipment. They must acknowledge, however, that vast areas remain to be conquered. Diseases of the joints are at once the most disabling and the most baffling of human complaints. Many years ago Osler wrote that *arthritis deformans* was an incurable disease, and he said that the best that could be hoped for was a gradual arrest of the condition. The man who today echoed Osler's views could not be justly accused of undue pessimism. It is salutary therefore to take stock of the present position, to see what can be done for sufferers and to determine lines for future research.

The Council of the British Medical Association has recently published in *The British Medical Journal* of June 17, 1933, a lengthy report on the causation and treatment of arthritis and allied conditions. *The British Medical Journal* is received by all readers of this journal who are members of the British Medical Association. There is thus no need to attempt a *précis* of the report, to recapitulate its origin, or to name the members of the committee responsible for it. There is need, however, for all medical practitioners in Australia to study the report and to read the leading article accompanying it. This is not a subject that has an interest peculiar to the United Kingdom. Arthritic disablement is world-wide and, as Australian practitioners know, is far too often seen in the Commonwealth. This report will clarify the ideas of Australian practitioners. It will doubtless also lend itself to discussion by those who have fixed ideas about ætiology or classification or treatment.

The question of classification and nomenclature, referred to in *The British Medical Journal's* leading article, though important in the differentiation of certain types of lesion, need not be discussed in this place. Rather would we refer to treatment. The report is concerned only with types of arthritis of which the ætiology is uncertain. At the same time, certain factors in the causation even of these types are known. The most important of the factors is infection, and the most usual sites for infection are teeth, tonsils, nasal sinuses, pharynx, intestines and genital tract. Endocrine imbalance and allergic sensitivity have also to be considered. Treatment of the arthritic condition, in common with that of many other lesions, must, of course, be directed at the causative agent. But the causative agent must not be the sole object of attack. The bodily condition of the patient, the soil in which the infective agent may operate, must be considered.

In the British Medical Association report is an interesting section on biochemistry; in this section the blood, the urine, the sweat and the gastric juice are discussed. Relatively little is known about the chemistry of the body in diseases of the joints. The statement is made in the report that among the biochemical investigations that should be undertaken in future research are: (i) calcium metabolism; (ii) liver function; (iii) the presence of (*a*) hæmolysins, (*b*) glutathione in the blood; and (iv) skin reactions and allergic manifestations. It is quite simple to say that when a medical practitioner has discovered a septic focus or other infective agent in a case of arthritis, he must not

be content with his discovery, but must investigate the patient from every point of view to discover, by biochemical or other methods, any predisposing condition that may exist. To do this is not simple. We do not know enough about the chemistry of the body; we know practically nothing about what we term the resistance of the body to infection. In the report under consideration prophylaxis is discussed in two small paragraphs; therapy of various kinds occupies several pages. Ideally the proportion should be reversed. Until further research is carried to a successful issue we must pay attention chiefly to so-called actual causes. In the treatment of any patient with an arthritic condition the ætiological factor must always be the guide to treatment. A most instructive paper on this aspect of the subject was published in this journal by C. B. Blackburn as long ago as November, 1925; after all these years it is still worthy of study. Finally, if medical practitioners are to be successful in the treatment of arthritis, they must be able to recognize the several types in their early stages.

Current Comment.

PRIMARY PNEUMOCOCCAL PERITONITIS.

THE condition known as primary pneumococcal peritonitis is one in which the peritoneal lesion is the only manifestation of the disease. Much discussion has raged round its pathogenesis. Divergent views are held. Some observers state that the peritoneal lesion arises as the result of a septicæmia; others hold that the organisms gain access to the peritoneum by passing through the intestinal walls. A third group holds that infection always takes place by way of the genital tract. This subject was discussed several years ago in these pages on more than one occasion in connexion with views expressed by McCartney and by several speakers at a meeting of the Royal Society of Medicine. It may be remembered that McCartney concluded that primary pneumococcal peritonitis affected only females and that the infection reached the peritoneum by the vagina.

In a recent communication on pneumococcal infections in infancy and childhood, J. W. S. Blacklock and K. J. Guthrie devote some attention to pneumococcal peritonitis.[1] In a series of 2,000 autopsies they found pneumococcal peritonitis 24 times. They have also studied bacteriologically 15 other cases that did not come to autopsy. Of the 39 cases, 27 appeared to be primary, while 12 were diagnosed as secondary to some other pneumococcal lesion. Of the cases that came to autopsy, 12 were primary; of these, eleven occurred in girls and one in a boy. Of the 27 children with primary pneumococcal peritonitis, 24 were girls, the youngest being two months of age and the oldest eleven years. Fifteen were between two and seven years of age, an age at which, as shown by McCartney and Fraser, contamination of the external genital organs in girls of the poorer type is likely to occur. Three of the primary cases occurred in boys. In two of these cases the diagnosis was a clinical one; attention may therefore be fixed on the one case in a boy that came to autopsy. In this instance the inflammatory reaction was most acute in the region of the caecum. No lesion could be found in the caecum or on histological examination in the appendix, which had previously been removed at operation.

In an attempt to throw light on the pathway of infection, Blacklock and Guthrie examined 33 swabs from the vagina of healthy children by intraperitoneal inoculation into mice of the broth in which the swabs had been soaked. In only one instance was a pneumococcus (Group IV) isolated. They point out that Woods found the same type of organism once only in 71 cases. They conclude that the pneumococcus is rare in the vagina in childhood. Blacklock and Guthrie typed the pneumococcus in 15 cases; in 13 instances the organism belonged to Type I, in two to Type II. In four of the Type I cases swabs were taken from the vagina and also from the throat. In only one instance did they isolate a Type I pneumococcus from the vagina, and in that instance the same type was also obtained from the throat swab. Their results, as they state, are inconclusive. In ten cases described by McCartney in 1923 the type of pneumococcus was the same in the peritoneum, the blood and the vagina. It is of interest also to note that Reginald Webster, writing in this journal in 1924, reported that, though he found a specifically agglutinable pneumococcus in the peritoneal cavity, he was unable on two attempts to isolate pneumococci from the vaginal secretions. Blacklock and Guthrie publish a table in which are set out the types of pneumococci reported by various authors in peritonitis. The cases number 72; 53 were of Type I, nine of Type II, one was of Type III, and nine were of Group IV.

The observations of Blacklock and Guthrie do not lead us far in elucidating the mystery of pneumococcal peritonitis. (We have made reference to only one part of their article.) They do show, although perhaps none doubted it, that McCartney was wrong when he stated that males were not affected and that the infection always gained access to the peritoneal cavity by way of the genital tract. The incompleteness of the observations of Blacklock and Guthrie regarding the types of pneumococcus suggest lines along which every case of pneumococcal peritonitis should be investigated. Only by the most complete observation can further light be thrown on the subject. Fraser has stated that the matter is now as fully established as

[1] *The Journal of Pathology and Bacteriology*, May, 1933.

it is likely to be. We should not be content to allow this statement to stand.

The subject cannot be allowed to pass without mention of views expressed by Fraser in 1931 and reiterated by him in his contribution to this year's "Medical Annual". He admits three avenues of infection: the female genital tract, the respiratory tract and the intestinal tract. From a local focus a general infection arises, a condition virtually of septicæmia, and the peritonitis is but a tissue reaction to the general infection. From the evolutionary point of view the peritoneal space originally developed as a protective space around the dangerous infective area of the intestinal tube. He describes peritonitis developing without an intraperitoneal focus of infection as a peritoneal (mesothelial) reaction secondary to the general disturbance. He adds that in this event the peritonitis is in some respects a salutary feature in so far as it affords possibilities of an immunity influence.

BILIARY STASIS.

THERE are times when every medical practitioner must wonder if he really belongs to a scientific profession. Such a doubt is not without foundation when we remember how striking advances have been rejected by the less keenly endowed minds of the profession. On what slender evidence do often we base our opinions! No wonder the "pure" scientists complain that much of our work lacks adequate control. But, passing by for the time being the not unjustified rejoinder that the chemist and physicist are not always immune from error or loose thinking, we must admit that our terminology often enslaves us. Of this the word "stasis" is an apt example. In the course of the Goulstonian lectures on the physiology of the gall-bladder, Charles Newman remarks that this word has been one of the Mesopotamias, a blessed word of great soothing, but often employed so loosely as to be not in concordance with fact.[1] For some time past it has been accepted that gall-stones are produced by many important causes, of which not the least important is stasis. This has been believed without a careful examination of just what is meant by this term with special reference to the physiology of the gall-bladder. Sir Thomas Lewis has recently pointed out how the words "compensation" and "decompensation" have more or less dominated our concepts of cardiac failure; "stasis" is a comparable term. Newman points out that stasis of bile in the gall-bladder is possibly a condition for the evolution of other processes. But he justly goes on to say that it is these other connecting links, the other processes that are the mediate causal agents between the slowing of the biliary movement in the extrahepatic channels and the genesis of disease, that are really of interest. To give stasis as a cause gives a basis for argument, for experiment, even for proof, but the word in itself is no explanation. We must agree with this author when he claims that in the strictest sense stasis means standing still, just as the contents of the gall-bladder would stand still if the cystic duct were tied. Consequently he calls this really static condition "standstill", such as would occur with obstruction of the cystic duct by growth, stone or fibrous tissue. How can this lead to stone formation? There are two possibilities: that the bile may undergo decomposition and that cellular *débris* and minute stones may thereby be retained and form nuclei for larger stones. As regards the latter, minute stones may readily be left behind in a normal gall-bladder, and cells apparently are not of importance as the nuclei of cholelithis. Newman states that since the selective abnormalities of concentration in the inflamed gall-bladder as regards bile salts are now better understood, there is no need to invoke stasis as a cause *per se*. He borrows an analogy from the language of the urologists and remarks that most of us go through life with a few cubic centimetres of residual bile. If this argument is admitted as sound, it will be seen that there is sufficient "stasis" in the average gall-bladder to favour stone formation once the process has started. Therefore we need not invoke even a less complete biliary traffic block than standstill as a major cause for gall-stones. The other contributing causes thus appear important.

Of course this does not mean that therapeutic measures designed to empty the gall-bladder as completely as may be are not indicated. It merely implies that we have made no explanation when we have said "stasis" and passed on content. Newman speaks also of distension of the gall-bladder, a process which is normal in the fasting or digesting individual. This condition gradually becomes pathological when it is unduly prolonged. But even then, all that could happen in an otherwise healthy organ would be undue concentration of bile, and thence precipitation. This reaction would, however, be a reversible one, and therefore of itself could not lead to gall-stone formation.

That true stasis in the gall-bladder may cause hydrops of the organ is, of course, true. Experimental and clinical evidence also points to its importance in causing cholecystitis. This brings us to the important infective factor, which, with the aberrations of cholesterol metabolism, are subjects too wide for discussion here. Newman's lectures also contain much interesting material anent the biliary dyskinesias, their types and their clinical recognition. But we can with advantage return to the point under present review and attempt to adjust our thinking to our knowledge of facts, with special regard to our medical currency of speech. To speak of a toxæmia is, after all, but hellenizing the idea of poison in the blood. To speak of cephalalgia is not to explain headache. Similarly, to invoke stasis as a mechanism for gall-stone formation is really not introducing novelty nor even an hypothesis. In other words, when we use special terms we should honestly ask ourselves just what we mean by them.

[1] *The Lancet*, April 29, 1933.

Abstracts from Current Medical Literature.

OPHTHALMOLOGY.

The Pituitary Thalamic Region and the Eye.

H. Zondek and G. Koehler (*Deutsche Medizinische Wochenschrift*, December 23, 1932) have closely investigated the relationship in disease between the pituitary thalamic region and the eye, for developmentally they are closely related. The Lawrence Biedl syndrome and the juvenile type of amaurotic familial idiocy may involve both these areas. The frequent occurrence of *retinitis pigmentosa* in the Lawrence Biedl syndrome led to the belief that it was due to pituitary disease, since the intermediate part of the pituitary produces a hormone closely related with pigment metabolism. In the authors' clinic they have noted in patients with thalamo-pituitary disease a greater percentage with eye disturbances such as astigmatism, hypermetropia, myopia, strabismus and *retinitis pigmentosa*. Even if patients with *retinitis pigmentosa* do not show definite endocrine anomalies, such as *dystrophia adiposo-genitalis*, one will often find on very careful examination a disturbance of the pituitary thalamic system (salt retention, lowered sugar tolerance, diminished specific dynamic action of food). The authors concluded that in some cases the degenerative process involved the floor and walls of the third ventricle and extended along the optic nerve to the eye, especially to the retina.

The Light Streak on the Retinal Vessels.

W. H. Wilmer, H. F. Pierce and J. S. Friedenwald (*Archives of Ophthalmology*, March, 1933) explain that, as the blood vessels are not self-luminous, the light streak must be a reflection of the light employed. If reflected from the chorioid, it would necessarily have a red colour, whereas it has the same colour as the light used. Even the vessels on a detached retina show the light streak in those regions in which the surface of the retina lies perpendicular to the line of view. Otherwise the streak is absent. The slit lamp shows the presence of the streak on vessels in the conjunctiva and cornea. Hence the chorioid may be excluded as the reflecting surface. Experiment with a glass tube filled with opaque solution shows that light reflected from behind is reflected by the vessel wall into two bright streaks, one on each side of the "blood column". Hence the surface behind the blood column can be excluded. Reflection can take place only in the surface between each successive pair of the following structures: vitreous, retina, adventitia, media, intima and blood. The surface vitreous-retina accounts for the brilliant reflexes that are seen about the macula, but parallactic movements show that they are in front of and independent of the vessel reflection. The retina-adventitia may be excluded also, as under ordinary circumstances the index of refraction of the adventitia is equal to that of the retina. The adventitia-media surface separates structures of widely different densities and reflection of light should therefore occur here. In pathological conditions, such as arteriosclerosis with thickening of the media, a glassy coat surrounding the artery, such as this theory predicates, is readily seen and is familiar to the ophthalmologist. The surface media-intima and intima-plasma are so close that they may be considered as one in the artery. Also the adventitia-intima and intima-plasma may be considered one in the vein. Most writers agree that reflection takes place at this surface, casually described as the surface. By an optical formula it may be worked out that the width of the light streak is one-twentieth of the diameter of the vessel. When the width of the reflex is greater than this, some other surface must have contributed, namely, the adventitia-media.

Vascularization of the Anterior Eye Segment.

P. C. Jameson (*Archives of Ophthalmology*, April, 1933) discovered in actual operative practice that excessive vascularization of the conjunctiva and cornea was derived not only from the conjunctival circulation, but also from the ciliary system. In many cases a conjunctival peridectomy was insufficient for any relief of the condition. When an area of conjunctiva was removed from the neighbourhood of the limbus, higher magnification showed a reticulum of deeper ciliary vessels covered by a glossy, resistive, translucent membrane, probably Tenon's capsule. It cannot be removed by dissection and protects the underlying vessels from destruction by a curette. The author attacks the vessels by numerous parallel linear incisions with a Graefe knife over a given area. He calls this procedure ciliarotomy and regards it as a useful operation in the temporary relief of acute glaucoma or in the alleviation of pain in blind glaucomatous eyes, or in other congestive conditions.

Fixation and Voluntary Nystagmus.

A. L. Luhr and J. L. Eckel (*Archives of Ophthalmology*, April, 1933) report that they failed to find any case of fixation nystagmus in English or American literature, and only a few in German. Voluntary nystagmus, though also rare, has been discussed for many years, and thirty cases are on record. A boy, aged thirteen years, otherwise healthy, discovered that for the last three years when he fixed an object the eyes would shake. It interfered with his reading, but happened also when he looked at distant objects. The oscillations were horizontal and rotary-horizontal with an amplitude of about one millimetre and a rate of between 400 and 500 per minute. They lasted a few seconds; then followed rapid convergence and painful closure of the lids. A trained nurse, aged twenty-one, from a small child would make her eyes "dance" at will. The oscillations were about 300 to the minute. She was a normal young woman. The explanations offered for these conditions are numerous and vague. "The sending of strong stimuli from the rhythmic centre of the cortex, producing alternating rather than simultaneous discharges of energy, is one explanation." Others think it is a fatigue process.

Retinal Detachment Treated by Diathermy.

E. F. King (*The British Journal of Ophthalmology*, May, 1933) reports that during the last three years, of 200 patients suffering from detachment of the retina treated by Gonin's method, 27·5% were cured; of thirty treated by the Guist operation—multiple trephine and caustic soda cautery—45% were cured. Diathermy was then tried by the method of Larsson, of Stockholm, its aim being to produce a diffuse area of chorioiditis without affecting the vitreous. The hold is localized by the simple expedient of marking at the limbus with India ink under direct ophthalmoscopy the meridian in which the hole is situated, and estimating in due diameters the distance of the hole in that meridian backwards. The mark is on the sclera. A subconjunctival and sub-Tenon's capsule injection of "Novocain" and adrenaline is given in the neighbourhood of the hole. The field of operation is exposed and cauterization is begun over the position of the hole. The electrode must be in contact with the sclera before the current is turned on; five seconds are allowed and the current is turned off before the electrode is removed. The procedure is to begin with a low current (0·75 ampère) to avoid perforating the sclera and chorioid. This is repeated over the required area, about 1·5 millimetre interval being allowed between each application. Finally, the subretinal fluid is evacuated from the region where the detachment is deepest, by perforating the sclera with the electrode or trephine and the chorioid by a blunt instrument. A vulcanite speculum should be used. Of the thirty-one cases, cures were obtained in eighteen, that is, 58%.

Glaucoma and Nævus Flammeus.

C. S. O'Brien and W. C. Porter (*Archives of Ophthalmology*, May, 1933) cite from the literature published from 1860 to the present day numerous cases of nævus of the face associated with glaucoma. Besides the facial nævus, lesions include cavernous angioma of the chorioid, detachment of the retina, enlargement of the eye and hæmangioma of the

meninges. The authors' case was that of a woman of thirty with a port wine discoloration extending from the scalp over the entire forehead and temporal region of the left side. The left side of the nose was involved, but the lower lid was normal. The caruncle and palpebral conjunctiva were unduly red. Her left eye was blind since birth. She complained also of convulsions, attacks of unconsciousness and heart trouble. The cornea was enlarged, measuring 12 by 13 millimetres. The pupil, 5·5 millimetres in diameter, reacted badly to light. Tension was 12 (Schiötz). There was a deep pathological cupping of the disk. The diagnosis was unilateral hydrophthalmos, with homolateral *nævus flammeus* of the face, epilepsy, mitral insufficiency and probable meningeal hæmangioma. Apparently the association exists only when the nævus involves the lids and perhaps other ocular structures. The glaucoma is most frequently of the infantile type, as in most cases the globe is enlarged. The increased tension is thought to be due to the excessive transudation through the thin-walled chorioidal veins, or from a plethoric condition of the chorioidal vessels. Cushing believes that the facial nævus tends to follow the distribution of the fifth nerve.

OTO-RHINO-LARYNGOLOGY.

Tonsillectomy.

E. WIRTH AND E. RENNO (*Deutsche Medizinische Wochenschrift*, March 3, 1933 found that of 2,766 patients subjected to tonsillectomy in the ear, nose and throat clinic of Heidelberg between 1920 and 1932, the majority ran a course without marked symptoms. Hæmorrhage occurred in 2% to 3%, elevation of temperature above 37·5° C. (99·5° F.) in 5·8%, and bronchopneumonia in 0·2% of the cases. Tonsillectomy does not belong to the dangerous operations and also not to the harmless ones. Two deaths occurred from hæmorrhage *plus* bronchopneumonia. In all cases prior to operation it is necessary to consider the general condition and the age of the patient, the risk of hæmorrhage, and whether the end-result of the operation will produce a sufficiently good result to warrant this risk.

Post-Diphtheritic Laryngeal Stenosis.

CURT H. KREIGER (*Archives of Otolaryngology*, January, 1933) reviews the literature and reports six cases of post-diphtheritic laryngeal stenosis. He concludes that post-diphtheritic stenosis of the larynx cannot be ascribed to any one cause. Errors in intubation or tracheotomy, prolonged retention of tubes, and diphtheritic infection are the chief causes, in the order named. Intubation by direct laryngoscopy is to be preferred, because its performance can be readily learned, trauma to the larynx is lessened, and at the time of intubation the larynx can be studied. Tubes should not be worn over two weeks; probably a shorter period is preferable. Tracheotomy is indicated if extubation is impossible at this time. Dilatation should be instituted early if decannulation is impossible, as the cicatrix is still soft and responds to dilatation more readily. Jackson's method of dilatation and occlusion is the procedure of choice whenever applicable. When continuous dilatation is necessary or desirable, the method of Iglauer seems most logical. Knowledge of the nature and extent of the stenosed area, which can be determined by direct and retrograde laryngoscopy and by Röntgenological studies, is necessary before treatment is instituted. As most of the cases of laryngeal stenosis follow diphtheria in children of preschool age, early immunization of the very young should be urged. The prognosis for ultimate functional recovery is favourable if the cartilaginous framework is not entirely destroyed. Treatment should be carried out in a well regulated hospital, where the patient is under the constant supervision of watchful nurses specially trained for this work.

The Tonsil Problem.

OTTO KAHLER (*The Journal of Laryngology and Otology*, January, 1933) discusses the physiological rôle of the tonsils and the question of so-called chronic tonsillitis, particularly in its relation to general affections. He agrees with Bergen and Heilman, who conclude that the tonsils must be considered as protective organs which, particularly during childhood, have the important function of keeping the body ready for defence. But as this function must be the same for the whole lymphoid tissue of the body, the loss of the tonsils, as an extremely small part of the lymphoid tissue of the body, must not be over-valued. It has not yet been possible to prove that the tonsillar tissue affects internal secretion. It is now generally recognized that rheumatic diseases, endocarditis, septic diseases and nephritis are often connected with tonsillar sepsis. If there are frequently relapsing anginas or peritonsillar abscesses, if there are general diseases that are caused by a focal infection and for which no other focus is to be found, the tonsils must be removed. Tonsillectomy can do no harm. In spite of all attempts to prove the contrary, this fact has been confirmed by clinical experience. It is an error to think that patients whose tonsils have been removed suffer more frequently from colds or infections than others. These illnesses cannot be entirely prevented by tonsillectomy, for they are diseases of the mucous membrane and are often due to the constitutional inferiority of the mucous membrane. The apparently greater frequency of colds that some patients complain of is easily explained by the fact that after tonsillectomy has been performed the diseases of the mucous membranes receive more attention, whilst before the operation the grave symptoms of the tonsillar disease claimed chief attention. The author condemns the so-called conservative treatment of chronic tonsillitis and favours radical removal.

Early or Late Operation in Acute Mastoiditis.

DOUGLAS GUTHRIE (*The Journal of Laryngology and Otology*, June, 1933) discusses the question of how early operation should be undertaken in cases of acute mastoiditis. The study of a series of cases appears to indicate that the second or third week of the otitis is the safest period. In the great majority of cases, that is, in mastoiditis of average severity, early operation is sound in practice and gives good results. In mild cases there is always the possibility of recovery without operation, and it should be laid down as a general working rule that early mastoid symptoms do not always demand immediate operation. In the very acute or so-called fulminating cases the author questions whether operation during the first week is as safe as some would have us believe. The author asks whether it may not aggravate or even induce a condition of septicæmia by removing the nucleus of a "fixation abscess" which is forming with the mastoid antrum and cells. He also asks whether a few days' delay may not be fully justified in certain cases of this nature. The author concludes that no rigid rule for all cases can be given regarding the most favourable time for operation. In each the choice calls for careful judgement and must always be a matter of individual opinion.

Irrigation of the Ear in Acute Otitis Media.

DAVID SHAPIRO (*Archives of Otolaryngology*, March, 1933) undertook to determine whether there is risk of spreading the infection to the mastoid cells in irrigation of the ear in acute *otitis media*. He selected a few patients with mastoiditis who required operation, and irrigated the ear with a 1% solution of methylene blue one hour prior to operation, using for this purpose 475 cubic centimetres (a pint) of fluid from a douche bag suspended 60 centimetres (two feet) above the level of the ear. In addition, 50 cubic centimetres of the same solution were injected into the ear while the patient was on the operating table, just before the primary incision was made. The canal was dried out thoroughly before the external plate of the mastoid was opened, and in not one instance was a trace of the dye found, even in the most anterior cells. Similar experiments were carried out on two cadavers. The author concludes that under ordinary circumstances irrigation of the ear is entirely safe so far as carrying infection back into the mastoid is concerned.

Special Articles on Treatment.

(Contributed by request.)

XV.

THE TREATMENT OF COLLES'S FRACTURE.

COLLES'S FRACTURE was described by Pouteau in 1783 and by Colles in 1814 as a transverse fracture of the distal end of the radius within about one inch of the inferior articular surface. Some forty years later Smith described the triple displacement of the distal fragment which has come to be regarded as a characteristic of the condition when displacement exists. The injury is usually caused by a forced hyperextension of the wrist, such as would occur by a fall on the outstretched hand, and is considered by some to be the result of this cross-breaking strain on the distal end of the radius. The continuation of the violence in most cases tends to force the lower fragment dorsally and in a radial direction, at the same time producing a deviation of the inferior articular surface of the radius, so that this surface, instead of making an angle of 85° with the axis of the radius in the sagittal plane and 30° in the coronal plane, faces either directly downwards or even downwards and backwards. Some writers consider that the essential feature of the displacement is a forward luxation of the lower end of the ulna, but this view is contested on the ground that the distal fragment of the radius really rotates about the head of the ulna, producing thus a relative forward luxation of it. There is some degree of displacement of the distal fragment in about 75% of all cases of Colles's fracture. Sometimes the displacement is minimal and the fragments are impacted, resulting in a buckling of the cortex on each aspect of the bone. With the exception of those cases in which the displacement is minimal, a hæmatoma rapidly obscures the deformity. When the displacement is extensive, the characteristic "dinner fork" deformity is present and the tendency of the distal fragment to be displaced radially results in rupture of the medial collateral ligament of the wrist joint or fracture of the styloid process of the ulna. Injury to the triangular fibro-cartilage rarely occurs. The periosteum on the anterior aspect of the radius is commonly torn, but that on the posterior aspect frequently escapes injury, except in the case of anterior displacement. The line of fracture is generally transverse and may involve the inferior radio-ulnar articulation, which, however, is very rarely involved if there is an associated fracture of the styloid process of the ulna. The distal fragment is frequently slightly comminuted and impacted, and even in these cases there is a tendency for the distal fragment to be tilted backwards on a transverse axis. In other cases characterized by the triple displacement the distal fragment may actually lie with its anterior surface in contact with the posterior surface of the proximal fragment and any intermediate degree of displacement may be observed. Crepitus may be absent owing to impaction of the fragments. In all except those cases with minimal displacement careful palpation will reveal a partial or complete obliteration of the anterior curve of the radius and an alteration in level of the radial styloid process as compared with that of the ulna; but the backward tilting of the distal fragment, which is one of the most important features of the displacement, may escape notice before careful scrutiny of the radiograph, which should always be taken in the antero-posterior and lateral planes.

Sometimes the fact that a small piece of bone has been separated from the anterior aspect of the lower end of the shaft will be the only evidence that an epiphyseal separation has occurred and that the deformity has been reduced either spontaneously or prior to radiographic examination.

Observation of the radiographs will reveal the characteristics of the displacement as described by Skinner. The inferior articular surface of the radius should normally make an angle of 30° with the axis of the bone in the coronal plane, whereas, owing to the radial displacement of the distal fragment in a Colles's fracture, this angle may even become 90°. Similarly, in a lateral view the backward tilting of the distal fragment increases the angle made by the inferior articular surface with the axis of the radius in the sagittal plane in comparison with the normal angle of about 85°.

Seeing that impaction is such a characteristic feature of this injury, the first essential in treatment is to secure complete relaxation of the muscles in order to disimpact the fragments. This relaxation may be secured by injecting a 2% "Novocain" solution between the fragments. Laming Evans has pointed out the necessity of injecting this "Novocain" into the hæmatoma which forms around the site of fracture, and when there is much displacement it is necessary to inject the "Novocain" into the posterior as well as the anterior portion of the forearm. This procedure finds its maximum usefulness in the early treatment of fractures, as it is much more difficult to secure perfect analgesia in the fragment when the hæmatoma has been absorbed. Needless to say, the greatest care should be taken to preserve asepsis.

It is generally agreed that narcosis by means of gas, or gas and oxygen, is of too short a duration to permit of adequate reduction and retention of this type of fracture; and when a general anæsthetic seems indicated, it is better to give ether by the open method, so that there may be time not only to reduce the fracture, but to apply any retentive apparatus, and subsequently to insure that reduction is maintained before the patient recovers from the anæsthetic.

The primary consideration in reduction of the deformity is to secure complete anatomical reposition of the fragments. Practically all authorities are now agreed that it is very unusual to secure a large percentage of perfect functional results in the absence of perfect anatomical replacement.

The manipulations necessary to secure this ideal are hyperextension at the wrist joint to disimpact the fragments followed by traction on the distal fragment while the wrist is carried into volar flexion, pronation and adduction. During this procedure the thumbs are used to correct the posterior displacement and tilting of the lower fragment, which it is obviously impossible to do unless there has been perfect disimpaction. It is occasionally necessary to use a wedge-shaped piece of wood, the edge of which is covered with rubber, as a fulcrum over which the fragments are forcibly flexed in order to break down impaction, but as a rule it is possible and advisable to depend on manipulation by the operator's hands only.

When difficulty is experienced in retaining the fragment in its corrected position the so-called Cotton-Loder position is of great value. The wrist is placed in the position of flexion and ulnar deviation, with the forearm pronated after complete reduction has been secured. In this position the radial displacement of the distal fragment, the dorsal displacement and the backward tilting are all corrected by traction on the carpal ligaments, and any lesion of the ulnar collateral ligament of the wrist joint is allowed to unite in the correct functional position.

When the above measures fail it is wise to make an incision over the fracture site between the tendons of the *extensor pollicis longus* and *extensor carpi-radialis brevis* muscles. Direct traction is exerted on the two fragments by means of hooks, the incision being closed after the fragments have been drawn into apposition.

It may be necessary to resort to skeletal traction in which a Kirschner wire is inserted through the heads of the metacarpals and another through the proximal end of the ulna for counter extension, but the cases which will not yield to less radical measures are very few and far between. The degree of reduction may be checked by comparing the relation of the styloid process of the radius to that of the ulna with similar points on the uninjured limb, and also by the ability to secure volar flexion to at least 90°, but a control radiograph is essential for accuracy.

Having reduced the deformity, it is necessary to fix the fragments and retain them in position. It cannot be emphasized too strongly that complete reduction must be obtained before any retentive apparatus is applied. When the displacement has been minimal, a strap of adhesive plaster, 6·25 to 7·5 centimetres (two and a half to three

inches) wide, extending around the lower portion of the forearm will be sufficient, or light splints of aluminium which are moulded to conform to the curves of the forearm. It is bad practice to use splints for fixation of this injury unless they are adequately moulded or padded to meet the anatomical condition. Particular attention should be paid to the preservation of the anterior curve of the lower end of the radius by means of a dorsal pad between the splint and distal fragment, and a volar pad between the splint and distal end of the proximal fragment, so that the carpal articular surface of the radius will occupy its normal plane after the fracture has united. If wood splints are used, they should not extend past the distal crease in the palm or the heads of the metacarpals on the dorsal aspect. They should be slightly wider than the forearm to avoid encroachment on the interosseous space; they should be padded to conserve the anterior curve of the radius and should be adequately fixed, preferably by strapping encircling the limb and splint, so that there will be no rotary movement between the splints and the limb, and the anterior splint should be cut away opposite the thenar eminence.

Many varieties of splints have been used for this injury, but no splint will obviate the necessity of meticulous attention to detail in achieving full anatomical reduction and maintaining correct position while securing early movement of thumb and fingers. Plaster of Paris is almost ideal in the treatment of these fractures owing to the fact that it may be so moulded to the limb as to maintain the inferior articular surface of the radius in its correct plane and at the same time to allow of free movement of the thumb and fingers. The anterior plaster slab as advised by some writers appears to be contraindicated owing to the fact that it fails to prevent recurrence of the deformity. The posterior plaster slab, however, will prevent recurrence of the deformity, provided it extends from the elbow to the heads of the metacarpals and is applied with the wrist in slight flexion. An encircling plaster of Paris dressing is of the greatest value owing to the fact that it may be left on longer than either the anterior or posterior slab, that it retains itself in position without any constricting dressing, and that it may be so moulded as to provide for full and free movements of the thumb and fingers.

The first essential in after-treatment is to maintain immobilization of the fragments of the radius while encouraging early active movements of the fingers and thumb. Opinion has been divided as to whether absolute rest of the fragments should be of long or short duration, but current practice favours immobilization for as short a period as possible. The dorsal half of the plaster or the posterior splint may generally be discarded in a week or ten days, but the anterior support should be retained for one and a half to two weeks longer. Folded newspaper or corrugated cardboard makes a very light but comfortable splint in this stage. During this latter period the splint is removed each day and massage is administered. Active movements of the wrist joint, fingers and thumb are encouraged, while the site of fracture is protected by the hand of the masseuse. Prolonged immobilization of the wrist joint is probably responsible for many of the bad results following this injury.

When the Cotton Loder position has been used, it must not be maintained for more than four or five days. Usually on the fourth day a horseshoe-shaped piece of plaster is removed over the dorsum of the hand and carpus and the limb is then turned over in supination so that gravity assists in producing dorsiflexion; after this exercise the hand is again tied down in the volar flexed plaster, being removed each day for active dorsiflexion. The plaster is replaced by a light anterior splint at the end of ten days.

If massage is given early, one must avoid any displacement of the distal fragment and any movement between the fragments. Consequently the anterior splint should not be removed for massage during the first ten days, and if this practice is adopted the chief objection to early massage should disappear. Extraarticular adhesions should, if possible, be prevented. Should they occur, diathermy is of value after they have been broken down.

One of the most common complications apart from malunion is stiffness and consequent loss of power of the fingers. This should be prevented by adequate and early movements of the fingers, which should be possible whatever retentive apparatus is used. Contusion of the articular cartilage of the shoulder joint owing to the violence of the original injury being transmitted through the bones of the arm and forearm is a not infrequent complication and manifests itself by pain commencing in the shoulder about three weeks after the injury, at which time proliferation of the connective tissue has produced a tender pannus over the damaged area of cartilage.

Arthritis in the inferior radio-ulnar joint is not a common complication, but does occur when reduction has been incomplete in those cases in which the fracture line involves this joint.

Injury to adjacent nerves is relatively rare; any of the nerves in the vicinity may be subjected to pressure, but rarely to laceration. A trophœdema of the hand, which occasionally follows these injuries, has been described as due to interference with the ganglion in which the posterior interosseous nerve ends on the dorsum of the carpus, but is probably due to prolonged immobilization and consequent vascular stasis. Occasionally the *extensor pollicis longus* tendon ruptures spontaneously some weeks after the injury. When this occurs it is due to attrition of the tendon where it passes over the rough edge of the partially reduced distal fragment. A stenosing teno-vaginitis affecting the tendons in the vicinity of the attachment of the brachio-radialis has also been described, but is not common, and an osteo-periostitis of the radius at this point may follow the displacement. Associated fractures of the carpal bones are rare, but when present the necessity for more prolonged immobilization complicates the treatment. Backward dislocation of the wrist associated with Colles's fracture is also rare, but presents no such complication in treatment. Preexisting arthritis in the wrist joint or in the inferior radio-ulnar articulation always complicates convalescence in this injury, and this possibility should be borne in mind when giving a prognosis. Malunion is probably one of the most important complications, as most writers are now agreed that where any displacement of the distal fragment persists after union a really good functional result is never attained.

Perfect anatomical reduction, entire absence of local tenderness, full function of fingers, thumb, wrist and radio-ulnar articulations constitute the criterion of a good end-result. This will be obtained only if the pathology of the condition is constantly borne in mind during treatment, and if treatment is carried out with due regard to prevention of the various complications. The patient, not the surgeon, will be the final arbiter of the value of the functional result.

R. V. GRAHAM, M.D., Ch.M. (Syd.), F.R.A.C.S.,
Honorary Assistant Orthopædic Surgeon, Lewisham Hospital; Honorary Assistant Medical Officer, Department of Orthopædics and Special Therapeutics, Sydney Hospital; Honorary Consulting Orthopædic Surgeon, Western Suburbs Hospital; Honorary Surgeon, New South Wales Masonic Hospital.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SECTION OF SURGERY OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Adelaide Hospital on March 8, 1933, DR. LEONARD C. E. LINDON in the chair. The meeting was devoted to a series of clinical and pathological demonstrations.

Recurrent Dislocation of the Shoulder Joint.

DR. IAN HAMILTON showed a healthy man, aged thirty-five years, who complained of having had a "fatty tumour" just in front of the left shoulder and of having dislocated

his left shoulder upwards of fifty times, so frequently and at such little provocation that he had developed a protective complex whereby he never allowed his left arm more than about 45° out from the side of the chest. When dislocated, his shoulder was easily reduced by taking hold of some firm object, for example, a door or table, and wobbling the shoulder, the bone slipping back into place.

Examination had revealed a lipoma about 10 centimetres (four inches) in diameter just anterior to the left shoulder joint. The joint itself did not show any physical abnormality.

On September 17, 1932, under ether anæsthesia induced by the open method, the lipoma was dissected away through a 12·5 centimetre (five inch) incision down the arm, commencing at the coracoid process. Then, through the same incision, the deltoid muscle was split along its anterior edge and the upper end of the humerus was exposed, part of the insertion of the *pectoralis major* muscle was cut away from the pectoral ridge of the humerus and the quadrilateral space was exposed and cleared. A long incision was then made over the back of the arm, the deltoid muscle was again exposed, and a sufficiently wide portion of it was separated from the rest of the muscle and stripped right down to the insertion, the nerve and blood supply being carefully preserved; this was then threaded through the quadrilateral space and gripped in front. The posterior wound was then closed. The muscle brought through was securely stitched with kangaroo tendon to the edges of the deltoid in front, the *pectoralis major* muscle was repaired, and the wound closed in layers.

Recovery was uneventful, the arm being tied to the side for six weeks, and on December 31 the patient wrote and reported a satisfactory result. He said that he could now swim overarm with complete confidence, which he had not been able to do for years.

When he was shown at the meeting, it was demonstrated that he could perform all movements with the affected arm as well as he could with the other, and he expressed perfect satisfaction with the result.

Hernia of the Linea Alba.

Dr. Hamilton's second patient was shown with the permission of Dr. Britten Jones. The patient was a stevedore, aged fifty-six, who complained that for the last two years he had found that whenever he worked he got a dull pain in the epigastrium, getting worse as the day went along, until after some hours' work he began to have a "heavy feeling" in the stomach, followed by weakness and giddiness, and sometimes accompanied by dry retching. This had been so severe that he had been forced to give up the last four jobs that he had secured. There was no relationship to the taking of food, and when he was not working he felt perfectly well; and as he had always been a very hard working man, he objected to idleness and wanted to work.

Examination revealed a fatty hernia of the *linea alba*, about 3·75 centimetres (one and a half inches) below the tip of the xiphoid process of the sternum. It was about the size of a pigeon's egg and had not been noticed by the patient. In addition, an opaque meal and shadowgraph showed that he had a duodenal diverticulum about the size of a large grape near the termination of the second portion of the duodenum.

The question which arose was whether the fatty hernia of the *linea alba* or the duodenal diverticulum was the cause of the peculiar symptoms, and on account of their being so definitely related to exercise Dr. Hamilton favoured the thesis that the hernia was responsible, probably pulling on the peritoneum and possibly on the *ligamentum falciformis*, and he proposed dealing with this first and later, if necessary, doing something for the duodenal diverticulum. The general consensus of opinion of the meeting favoured this course.

Defect in the Hard Palate.

DR. G. H. BURNELL showed a patient in whom he had repaired a defect in the hard palate by means of a pedicled tube graft. This case will be reported in a subsequent issue.

Mesenteric Thrombosis.

Dr. Burnell also showed a man, aged twenty-seven years, who was admitted to the Adelaide Hospital on February 16, 1933, with the following history. He had been well until five days before admission, when he was suddenly seized with severe pain in the lower part of the abdomen, more especially on the left side. The pain doubled him up and he vomited undigested food. He vomited again several times prior to admission. For the two days before admission he had had frequent bowel actions, passing mucus, but no blood, and he had been troubled by a frequent desire to pass urine. He had been a medical in-patient two weeks before this admission, suffering from Vincent's angina.

On examination his temperature was 37·9° C. (100·2° F.), his pulse rate was 110, and that of his respiration 20. His face was drawn, and he was obviously in pain. A general examination revealed no abnormality except in the abdomen. The abdomen was slightly distended, moderately rigid all over, and tender in both iliac fossæ. There was some dulness in both flanks. A rectal examination revealed no swelling. A diagnosis of probable pelvic appendicitis was made, and the abdomen was opened a few hours later. On opening the peritoneal cavity, a large amount of clear, straw-coloured fluid escaped; this was not blood-stained. An examination of the appendix showed it to be congested, but it was obviously not the cause of the patient's illness. Further exploration revealed a firm mass lying just below the pelvic brim, and on delivering this it was seen to consist of the last twenty centimetres (eight inches) of the ileum. It was dark red, but not plum-coloured, with a smooth surface, and with a few flakes of lymph adherent to it. The gut felt very firm and had the consistency of india-rubber. This condition of the gut ended quite abruptly above, where there was normal bowel. Passing from the coil of affected gut up to the root of the mesentery, there was a wedge-shaped mass of inflammatory tissue distending the mesentery, in which thrombosed veins could be readily seen.

A diagnosis of thrombosis of the mesenteric vessels was then made, and the question arose as to what was to be done with the loop of affected bowel. In view of the fact that the pathological condition had been in existence for five days and that the bowel had retained its vitality for that time, it was decided to do no resection, and after removing the appendix and swabbing the peritoneal cavity as dry as possible, the abdomen was closed without drainage.

The patient made an uninterrupted recovery and was discharged on March 12, 1933.

Palmar Ganglion.

DR. P. S. MESSENT showed a man, aged forty-eight years, on whom he had operated for a compound palmar ganglion.

He was admitted to hospital on November 10, 1932, complaining of swelling of the left wrist, which had been present for two and a half to three years, gradually increasing in size, and for the past few weeks had been very painful. The patient had difficulty in grasping objects with the left hand; he used a crutch on the left side, owing to old infective arthritis of the left hip joint.

His general health had been excellent; there had been no loss of weight. Examination revealed a well developed man. His colour was good. His heart and lungs were normal.

On the anterior aspect of the hand and forearm was a prominent swelling, including the whole of the palm of the hand and extending along thumb, index finger and little finger. The swelling was constricted by the anterior annular ligament and extended about half way up the anterior surface of the forearm.

Fluctuation was present and was transmitted from the palm to the swelling in the forearm; also from the radial to the ulnar bursa. Coarse crepitus was present on flexing the fingers.

Movements of the wrist were fairly good, but the finger movements were grossly limited and caused some pain.

On November 22, 1932, an operation was performed under ether anæsthesia. A tourniquet was applied and a longitudinal incision was made from the mid-point of the

forearm to the distal transverse crease of the palm. The anterior annular ligament was divided; the main mass of the synovial sheath was then dissected free, opened and dissected off the tendons. Several ounces of "melon seed bodies" were contained in the sheath. Some of the tendons, especially that of the *flexor longus pollicis*, were "shaggy and frayed out". The wound was closed without drainage and a plaster of Paris splint was applied.

Active movements of the fingers were commenced next day and were gradually increased. On December 20, 1932, the patient was discharged to the out-patient department with the wound firmly healed, and no swelling of the hand and forearm; movements of the wrist were complete, and the hand grip was fairly strong. All the fingers could be completely flexed except the thumb. The *flexor longus pollicis* was very weak.

Dr. Messent considered this case of sufficient interest to be shown as an indication of the results that can be obtained in this condition by the removal of the diseased tissue followed by active exercises.

DR. L. C. E. LINDON said that he had seen some good results in cases treated in this way by Sir Henry Newland.

DR. L. O. BETTS stated that Kanavel had reported a successful series of cases thus treated.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Balzer, Noel Francis, M.B., B.S., 1931 (Univ. Sydney), Tallimba.

Bye, William Alick, M.B., Ch.B., 1923 (Univ. Sydney), 143, Macquarie Street, Sydney.

English, James Cameron, M.B., B.S., 1931 (Univ. Sydney), Gunnedah.

Furner, Curzon Watkin, M.B., B.S., 1931 (Univ. Sydney), Newcastle Hospital, Newcastle.

Ping, Aubrey Moore, M.B., 1929 (Univ. Sydney), 43, Hall Street, Bondi.

Correspondence.

ANÆSTHESIA WITH PRENARCOSIS BY MORPHINE AND PARALDEHYDE.

SIR: In these days of "Avertin", "Luminal", "Sodium "Luminal", "Nembutal" and numerous other much lauded, though recently discovered, narcotizing agents, it is refreshing to have attention redirected to paraldehyde, which is cheap, easy to prepare for administration, and whose reputation has stood the test of time.

I refer to the very excellent paper by Dr. Garrett and Dr. Gutteridge on "Anæsthesia with Prenarcosis by Morphine and Paraldehyde" published in your issue of July 8.

About two years ago, emboldened by the example of Dr. G. V. Doyle, of St. Arnaud, I commenced giving rectal injections of paraldehyde in saline following a dose of morphine an hour or so before operation. At that time the only reference I could find in the literature as to the toxicity and fatal dose of paraldehyde was in Taylor's "Medical Jurisprudence" (eighth edition), where it stated that "thirteen drachms and three and a half ounces have been given without fatal effect, but, on the other hand, two ounces have proved fatal". Consequently, being somewhat alarmed by the depth and length of the pre-operative sleep in two patients, I wrote to Professor Osborne, of Melbourne, for guidance as to the pharmacology of paraldehyde and to references to this drug in the literature. However, he was not able to give me much satisfaction in regard to this particular use of the drug.

Since then there have been several articles in the various journals, but even before I saw the first of them I had ceased to worry over the very prolonged post-operative sleep, which sometimes lasted for twelve hours, and in one case for eighteen hours.

Morphine, one-quarter grain, and hyoscine, one-hundredth grain, are given two hours before operation and half an hour later paraldehyde (a drachm for each stone of body weight for an adult, with a maximum of eight drachms) mixed with seven times its quantity of saline is slowly injected into the rectum, which has previously been washed out. Immediately after operation the bowel is washed out again.

The dose of paraldehyde for children should be only from half to three-quarters of a drachm per stone of body weight; but women require the same dose as men, which is contrary to the experience of Dr. Garrett and Dr. Gutteridge.

May I ask these gentlemen why they infer (in their summary) that morphine and paraldehyde are useful as a preliminary to a short anæsthesia, but not (I take it) to a long one, and why these drugs are unsuitable in cases of pulmonary fibrosis or coronary sclerosis? And have they observed the fall in blood pressure to take place before the operation, or rather the operative anæsthesia, commenced? And, finally, do they really regard the prenarcosis as being responsible for the single fatality in their series of cases, or even as an accessory to the fact?

The cautiousness of Dr. Garrett and Dr. Gutteridge is not shared by G. Duke Stewart, who records his experience of 500 cases in *The British Medical Journal* (December 24, 1932), nor by T. A. B. Harris (*The British Medical Journal*, January 14, 1933) and many others. Nevertheless their paper is very much to be commended.

Yours, etc.,

EDWARD J. HANLY.

Birchip,
Victoria,
Undated.

Proceedings of the Australian Medical Boards.

TASMANIA.

THE undermentioned have been registered according to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Bryan, Francis John, M.B., B.S., 1928 (Univ. Melbourne), Public Hospital, Launceston.

Cotter, Timothy John, M.B., B.S., 1924 (Univ. Melbourne), Public Hospital, Launceston.

VICTORIA.

The undermentioned has been registered according to the provisions of the *Medical Act*, 1928, of Victoria, as a duly qualified medical practitioner:

Freeman, Margherita Mahy, M.B., Ch.M., 1911 (Univ. Sydney), 52, Collins Street, Melbourne, C.1.

Additional diplomas registered:

Edison, Milton Gray, M.D., 1933 (Univ. Melbourne).

Williams, Stanley William, M.D., 1933 (Univ. Melbourne).

Melville, Charles Bernays, F.R.C.S., 1931 (England).

Murray, Hugh Dynnyrne Macinnes Lathrop, D.P.H., 1930 (England).

Sayle, Thomas Osler, F.R.C.S., 1931 (England).

Farmer, Paul Ward (Junior), M.R.C.P., 1932 (London).

Rabinov, Louis, D.G.O., 1930 (Dublin).

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

READERS are reminded that it has been decided to include a Section of Anæsthesia in the Fourth Session of the Australasian Medical Congress (British Medical Association), to be held at Hobart on January 15 to 20, 1934. Those desiring to read papers are requested to notify either of the Honorary Secretaries before October 1, 1933. The Joint Honorary Secretaries of the Section are Dr. Cedric Duncombe, 180, Macquarie Street, Hobart, and Dr. Geoffrey Kaye, 14, Collins Street, Melbourne, C.1.

Books Received.

THE DIAGNOSIS AND TREATMENT OF DIABETES AND THE USE OF HIGH CARBOHYDRATE DIETS, by W. W. Ingram, M.D., and G. V. Rudd, M.Sc., with a preface by C. G. Lambie, M.D., F.R.C.P., F.R.S.E.; 1933. Australia: Angus and Robertson, Limited. Crown 8vo., pp. 26. Price: 5s. net.

HOW YOU WORK: AN INTRODUCTION TO THE HUMAN BODY, by I. Wilson, M.D.; 1933. Australia: Angus and Robertson, Limited. Crown 8vo., pp. 185, with illustrations. Price: 3s. 6d. net.

HOW YOU BEGAN: A CHILD'S INTRODUCTION TO BIOLOGY, by A. Williams-Ellis, with preface by J. B. S. Haldane; 1933. Australia: Angus and Robertson, Limited. Crown 8vo., pp. 91, with illustrations. Price: 2s. 6d. net.

Medical Appointments.

Dr. W. J. Pannell (B.M.A.) has been appointed Medical Officer of Health by the Wongan-Ballidu Road Board, Western Australia.

* * *

Dr. R. M. Dunstone (B.M.A.) has been appointed Government Medical Officer at Bogan Gate, New South Wales.

* * *

Dr. R. M. McMaster (B.M.A.) has been temporarily appointed Medical Superintendent, Office of the Director-General of Public Health, New South Wales.

* * *

Dr. H. V. D. Baret (B.M.A.) has been appointed Medical Superintendent, Office of the Director-General of Public Health, New South Wales.

* * *

Dr. H. W. Palmer (B.M.A.) has been appointed Medical Superintendent, Office of the Director-General of Public Health, New South Wales.

* * *

Dr. D. Wallace (B.M.A.) has been appointed Medical Superintendent, Office of the Director-General of Public Health, New South Wales.

* * *

Dr. H. K. Denham (B.M.A.) has been appointed Director, Tuberculosis Division, and Medical Superintendent, Office of the Director-General of Public Health, New South Wales.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", page xiv.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officers.

OTAGO HOSPITAL BOARD, DUNEDIN, NEW ZEALAND: Resident Medical Officer.

REPATRIATION COMMISSION, WESTERN AUSTRALIAN BRANCH OFFICE: Medical Officer.

ROYAL COLLEGE OF SURGEONS, LONDON, ENGLAND: Conservator of Museum and Director of Research.

VAUCLUSE INFECTIOUS DISEASES HOSPITAL, HOBART, TASMANIA: Matron.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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